

**IN THE UNITED STATES DISTRICT COURT
FOR THE WESTERN DISTRICT OF PENNSYLVANIA**

MADISON M. LARA, et al.,

Plaintiffs,

v.

COL. ROBERT EVANCHICK, Commissioner
of Pennsylvania State Police,

Defendant.

Civil Action No.:
2:20-cv-01582

**LIST OF EXHIBITS TO BRIEF OF AMICI CURIAE GIFFORDS LAW CENTER TO
PREVENT GUN VIOLENCE AND CEASEFIRE PENNSYLVANIA EDUCATION FUND**

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EXHIBIT 1

Maturation of the adolescent brain

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Abstract: Adolescence is the developmental epoch during which children become adults – intellectually, physically, hormonally, and socially. Adolescence is a tumultuous time, full of changes and transformations. The pubertal transition to adulthood involves both gonadal and behavioral maturation. Magnetic resonance imaging studies have discovered that myelinogenesis, required for proper insulation and efficient neurocybernetics, continues from childhood and the brain's region-specific neurocircuitry remains structurally and functionally vulnerable to impulsive sex, food, and sleep habits. The maturation of the adolescent brain is also influenced by heredity, environment, and sex hormones (estrogen, progesterone, and testosterone), which play a crucial role in myelination. Furthermore, glutamatergic neurotransmission predominates, whereas gamma-aminobutyric acid neurotransmission remains under construction, and this might be responsible for immature and impulsive behavior and neurobehavioral excitement during adolescent life. The adolescent population is highly vulnerable to driving under the influence of alcohol and social maladjustments due to an immature limbic system and prefrontal cortex. Synaptic plasticity and the release of neurotransmitters may also be influenced by environmental neurotoxins and drugs of abuse including cigarettes, caffeine, and alcohol during adolescence. Adolescents may become involved with offensive crimes, irresponsible behavior, unprotected sex, juvenile courts, or even prison. According to a report by the Centers for Disease Control and Prevention, the major cause of death among the teenage population is due to injury and violence related to sex and substance abuse. Prenatal neglect, cigarette smoking, and alcohol consumption may also significantly impact maturation of the adolescent brain. Pharmacological interventions to regulate adolescent behavior have been attempted with limited success. Since several factors, including age, sex, disease, nutritional status, and substance abuse have a significant impact on the maturation of the adolescent brain, we have highlighted the influence of these clinically significant and socially important aspects in this report.

Keywords: myelinogenesis, neurocircuitry, molecular imaging, drug addiction, behavior, social adjustment

Introduction

Significant progress has been made over the last 25 years in understanding the brain's regional morphology and function during adolescence. It is now realized that several major morphological and functional changes occur in the human brain during adolescence.¹ Molecular imaging and functional genomics studies have indicated that the brain remains in an active state of development during adolescence.¹ In particular, magnetic resonance imaging (MRI) studies have discovered that myelinogenesis continues and the neurocircuitry remains structurally and functionally vulnerable to significant increases in sex hormones (estrogen, progesterone, and testosterone) during

puberty which, along with environmental input, influences sex, eating, and sleeping habits. Particularly significant changes occur in the limbic system, which may impact self-control, decision making, emotions, and risk-taking behaviors. The brain also experiences a surge of myelin synthesis in the frontal lobe, which is implicated in cognitive processes during adolescence.¹

Brain maturation during adolescence (ages 10–24 years) could be governed by several factors, as illustrated in Figure 1. It may be influenced by heredity and environment, prenatal and postnatal insult, nutritional status, sleep patterns, pharmacotherapy, and surgical interventions during early childhood. Furthermore, physical, mental, economical, and psychological stress; drug abuse (caffeine, nicotine, and alcohol); and sex hormones including estrogen, progesterone, and testosterone can influence the development and maturation of the adolescent brain. MRI studies have suggested that neurocircuitry and myelinogenesis remain under construction during adolescence because these events in the central nervous system (CNS) are transcriptionally regulated by sex hormones that are specifically increased during puberty.

Neurobehavioral, morphological, neurochemical, and pharmacological evidence suggests that the brain remains under construction during adolescence,^{1,2,3,7,12,21,22,23,27,49} as illustrated in Figure 2. Thus, the consolidation of neurocybernetics

occurs during adolescence by the maturation of neurocircuitry and myelination. Although tubulinogenesis, axonogenesis, and synaptogenesis may be accomplished during prenatal and immediate postnatal life, myelinogenesis remains active during adolescent life. Neurochemical evidence suggests that glutamatergic neurotransmission is accomplished during prenatal and immediate postnatal life while gamma-aminobutyric acid (GABA)ergic neurotransmission, particularly in the prefrontal cortex, remains under construction during adolescence.² Hence, delayed development of GABAergic neurotransmission is held responsible for neurobehavioral excitement including euphoria and risk-taking behavior, whereas dopaminergic (DA)ergic neurotransmission, particularly in the prefrontal area, is developmentally regulated by sex hormones and is implicated in drug-seeking behavior during adolescence;³ thus, brain development in critical areas is an ongoing process during adolescence. Indeed, adolescents are risk-taking and novelty-seeking individuals and they are more likely to weigh positive experiences more heavily and negative experiences less so than adults. This behavioral bias can lead to engagement in risky activities like reckless driving, unprotected sex, and drug abuse.^{1–3} In fact, most drug addictions initiate during adolescence, and early drug abuse is usually associated with an increased incidence of physical tolerance and dependence. The hormonal changes

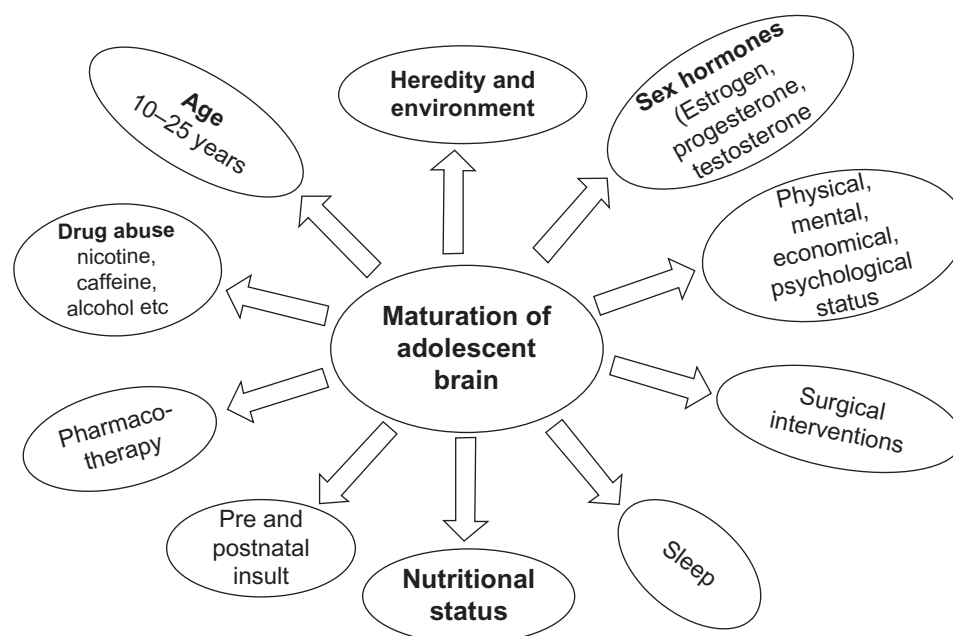


Figure 1 Factors influencing adolescent brain maturation.

Notes: Brain maturation is influenced by heredity and environment, prenatal and postnatal insult, nutritional status, sleep patterns, pharmacotherapy, and surgical interventions during early childhood. Furthermore, physical, mental, economical, and psychological stress; drug abuse (caffeine, nicotine, and ethanol); and sex hormones, including estrogen, progesterone, and testosterone influence the development and maturation of the adolescent brain. MRI studies have suggested that neurocircuitry and myelinogenesis remain under construction during adolescence because these events in the CNS depend on sex hormones that are specifically increased during puberty.

Abbreviations: CNS, central nervous system; MRI, magnetic resonance imaging.

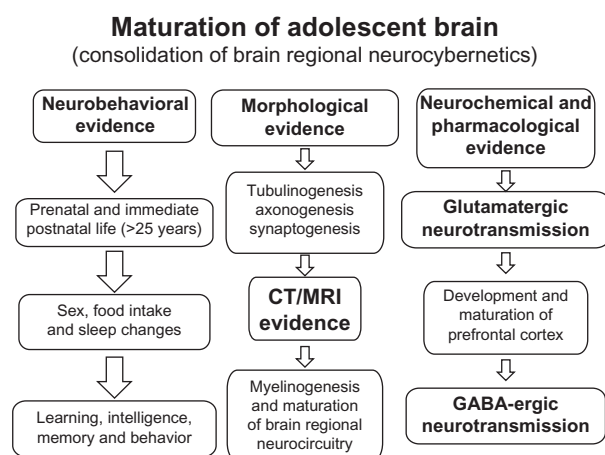


Figure 2 A diagram illustrating various stages of human brain development.

Notes: Several neurobehavioral, morphological, neurochemical, and pharmacological evidences suggest that the brain remains under construction during adolescence.^{1,2,3,7,12,21,22,23,25,42} Tubulinogenesis, axonogenesis, and synaptogenesis may be accomplished during prenatal and immediate postnatal life, yet myelinogenesis remains active during adolescent life. Furthermore, glutamatergic neurotransmission is accomplished during prenatal and immediate postnatal life, while GABAergic neurotransmission in the prefrontal cortex remains under construction. Delayed development of GABAergic neurotransmission among adolescents is implicated in neurobehavioral excitement and risk-taking behavior.

Abbreviations: CT, computed tomography; GABAergic, gamma amino butyric acid ergic; MRI, magnetic resonance imaging.

in puberty contribute to physical, emotional, intellectual, and social changes during adolescence. These changes do not just induce maturation of reproductive function and the emergence of secondary sex characteristics, but they also contribute to the appearance of sex differences in nonreproductive behaviors. Physical changes, including accelerated body growth, sexual maturation, and development of secondary sexual characteristics occur simultaneously along with social, emotional, and cognitive development during adolescence. Furthermore, the adolescent brain evolves its capability to organize, regulate impulses, and weigh risks and rewards; however, these changes can make adolescents highly vulnerable to risk-taking behavior. Thus, brain maturation is an extremely important aspect of overall adolescent development, and a basic understanding of the process might aid in the understanding of adolescent sexual behavior, pregnancy, and intellectual performance issues.

There are several other crucial developmental aspects of adolescence that are associated with changes in physical, cognitive, and psychosocial characteristics, as well as with attitudes toward intimacy and independence, and these may also influence brain maturation; these will also be discussed in the present report. Furthermore, we emphasize the deleterious effects of drug abuse and the clinical significance of nutrition from fish oils and fatty acids in adolescent brain maturation.

Neuronal plasticity and neurocircuitry

The term “plasticity” refers to the possible significant neuronal changes that occur in the acquisition of new skills.^{1–3} These skills initiate the process of elaboration and stabilization of synaptic circuitry as part of the learning process. Plasticity permits adolescents to learn and adapt in order to acquire independence; however, plasticity also increases an individual’s vulnerability toward making improper decisions because the brain’s region-specific neurocircuitry remains under construction, thus making it difficult to think critically and rationally before making complex decisions. Moreover, the neurocircuitry may be forged, refined or weakened, and damaged during plasticity. Thus, neuronal proliferation, rewiring, dendritic pruning, and environmental exposure are important components of brain plasticity during adolescence. A significant portion of brain growth and development occurring in adolescence is the construction and strengthening of regional neurocircuitry and pathways; in particular, the brain stem, cerebellum, occipital lobe, parietal lobe, frontal lobe, and temporal lobe actively mature during adolescence. The frontal lobes are involved in movement control, problem solving, spontaneity, memory, language, initiation, judgment, impulse control, and social and sexual behavior. Furthermore, the prefrontal cortex, which is implicated in drug-seeking behavior, remains in a process of continuous reconstruction, consolidation, and maturation during adolescence.

The adolescent brain

It is well established that various morphological and physiological changes occur in the human brain during adolescence. The term “adolescence” is generally used to describe a transition stage between childhood and adulthood. “Adolescence” also denotes both teenage years and puberty, as these terms are not mutually exclusive. The second surge of synaptogenesis occurs in the brain during the adolescent years. Hence, adolescence is one of the most dynamic events of human growth and development, second only to infancy in terms of the rate of developmental changes that can occur within the brain. Although there is no single definition of adolescence or a set age boundary, Kaplan⁴ has pointed out that puberty refers to the hormonal changes that occur in early youth, and adolescence may extend well beyond the teenage years. In fact, there are characteristic developmental changes that almost all adolescents experience during their transition from childhood to adulthood. It is well established that the brain undergoes a “rewiring” process that is not complete until approximately 25 years of age.⁵ This discovery

has enhanced our basic understanding regarding adolescent brain maturation and it has provided support for behaviors experienced in late adolescence and early adulthood. Several investigators consider the age span 10–24 years as adolescence, which can be further divided into substages specific to physical, cognitive, and social–emotional development.^{5,6} Hence, understanding neurological development in conjunction with physical, cognitive, and social–emotional adolescent development may facilitate the better understanding of adolescent brain maturation, which can subsequently inform proper guidance to adolescents.⁷

Longitudinal MRI studies have confirmed that a second surge of neuronal growth occurs just before puberty.^{1,7} This surge is similar to that noticed during infancy and consists of a thickening of the grey matter. Following neuronal proliferation, the brain rewires itself from the onset of puberty up until 24 years old, especially in the prefrontal cortex. The rewiring is accomplished by dendritic pruning and myelination. Dendritic pruning eradicates unused synapses and is generally considered a beneficial process, whereas myelination increases the speed of impulse conduction across the brain's region-specific neurocircuitry. The myelination also optimizes the communication of information throughout the CNS and augments the speed of information processing. Thus, dendritic pruning and myelination are functionally very important for accomplishing efficient neurocybernetics in the adolescent brain.

During adolescence, the neurocircuitry strengthens and allows for multitasking, enhanced ability to solve problems, and the capability to process complex information. Furthermore, adolescent brain plasticity provides an opportunity to develop talents and lifelong interests; however, neurotoxic insult, trauma, chronic stress, drug abuse, and sedentary lifestyles may have a negative impact during this sensitive period of brain maturation.^{8,9}

Out of several neurotransmitters in the CNS, three play a significant role in the maturation of adolescent behavior: dopamine, serotonin, and melatonin.^{3,8,9} Dopamine influences brain events that control movement, emotional response, and the ability to experience pleasure and pain. Its levels decrease during adolescence, resulting in mood swings and difficulties regulating emotions. Serotonin plays a significant role in mood alterations, anxiety, impulse control, and arousal. Its levels also decrease during adolescence, and this is associated with decreased impulse control. Lastly, melatonin regulates circadian rhythms and the sleep–wake cycle. The body's daily production of melatonin increases the requirement for sleep during adolescence.^{8,9}

Behavioral problems and puberty

It is now known that hormones are not the only explanation for erratic adolescent behavior; hence, investigators are now trying to establish the exact nature of the interrelationship between pubertal processes and adolescent brain maturation. Dahl has explained three main categories of brain changes related to puberty: (1) changes that precede puberty; (2) changes that are the consequence of puberty; and (3) changes that occur after puberty is over.⁹ The timing of these changes may underlie many aspects of risk-taking behavior. These changes, which are the consequence of puberty, occur primarily in the brain regions closely linked to emotions, arousal, motivation, as well as to appetite and sleep patterns. Brain changes independent of puberty are those related to the development of advanced cognitive functioning.

Animal studies have shown that sex hormones (estrogen, progesterone, and testosterone) are critically involved in myelination.¹² These studies have provided a relationship between sex hormones, white matter, and functional connectivity in the human brain, measured using neuroimaging. The results suggest that sex hormones organize structural connections and activate the brain areas they connect. These processes could underlie a better integration of structural and functional communication between brain regions with age. Specifically, ovarian hormones (estradiol and progesterone) may enhance both corticocortical and subcortical functional connectivity, whereas androgens (testosterone) may decrease subcortical functional connectivity but increase the functional connectivity between subcortical brain areas. Therefore, when examining brain development and aging, or when investigating the possible biological mechanisms of neurological diseases, the contribution of sex hormones should not be ignored.¹⁰

A recent study has described how the social brain develops during adolescence.¹⁰ Adolescence is a time characterized by change – hormonally, physically, psychologically, and socially. Functional MRI studies have demonstrated the developmental changes that occur during adolescence among white matter and grey matter volumes in regions within the “social brain.”^{1,7,12} Activity in the mesolimbic brain regions also showed changes between adolescence and adulthood during social cognition tasks. A developmental clock – along with the signals that provide information on somatic growth, energy balance, and season of the year – times the awakening of gonadotropin-releasing hormone (GnRH) neurons at the onset of puberty. High-frequency GnRH release results in the disinhibition and activation of GnRH neurons at the onset

of puberty, leading to gametogenesis and an increase in sex hormone secretion. Sex hormones and adrenocorticotrophic hormones both remodel and activate neurocircuits during adolescent brain development, leading to the development of sexual salience of sensory stimuli, sexual motivation, and expression of copulatory behavior. These influences of hormones on reproductive behavior depend on changes in the adolescent brain that occur independently of gonadal maturation. Reproductive maturity is therefore the product of developmentally timed, brain-driven, and recurrent interactions between steroid hormones and the adolescent nervous system.^{11,12}

Limbic system

The limbic system is a group of structures located deep within the cerebrum. It is composed of the amygdala, the hippocampus, and the hypothalamus. These brain regions are involved in the expression of emotions and motivation, which are related to survival. The emotions include fear, anger, and the fight or flight response. The limbic system is also involved in feelings of pleasure that reward behaviors related to species survival, such as eating and sex. In addition, the limbic system regulates functions related to memory storage and retrieval of events that invoke a strong emotional response. Neuroimaging studies have revealed that when interacting with others and making decisions, adolescents are more likely than adults to be swayed by their emotions.^{12–16} In addition, adolescents often read others' emotions incorrectly. These studies involved comparing a teen brain to an adult brain determined that adolescents' prefrontal cortices are used less often during interpersonal interactions and decision making than their adult counterparts. In fact, adolescents relied more on the emotional region of their brains when reading others' emotions, which is more impulsive when compared to a logical or measured interpretation. Thus, an understanding of how the limbic system and the prefrontal cortex are used has provided a partial explanation for certain characteristics of adolescents and adolescent behaviors, such as quickness to anger, intense mood swings, and making decisions on the basis of "gut" feelings. Because adolescents rely heavily on the emotional regions of their brains, it can be challenging to make what adults consider logical and appropriate decisions, as illustrated in Figure 3.

Prefrontal cortex

Recently, investigators have studied various aspects of the maturation process of the prefrontal cortex of adolescents.^{17,18} The prefrontal cortex offers an individual the capacity to

exercise good judgment when presented with difficult life situations. The prefrontal cortex, the part of the frontal lobes lying just behind the forehead, is responsible for cognitive analysis, abstract thought, and the moderation of correct behavior in social situations. The prefrontal cortex acquires information from all of the senses and orchestrates thoughts and actions in order to achieve specific goals.

The prefrontal cortex is one of the last regions of the brain to reach maturation, which explains why some adolescents exhibit behavioral immaturity. There are several executive functions of the human prefrontal cortex that remain under construction during adolescence, as illustrated in Figures 3 and 4. The fact that brain development is not complete until near the age of 25 years refers specifically to the development of the prefrontal cortex.¹⁹

MRI studies have discovered that developmental processes tend to occur in the brain in a back-to-front pattern, explaining why the prefrontal cortex develops last. These studies have also shown that teens have less white matter (myelin) in the frontal lobes compared to adults, and that myelin in the frontal lobes increases throughout adolescence.^{1,7,21} With more myelin comes the growth of important neurocircuitry, allowing for better flow of information between brain regions.^{20,21} These findings have led to the concept of frontalization, whereby the prefrontal cortex develops in order to regulate the behavioral responses initiated by the limbic structures. During adolescence, white matter increases in the corpus callosum, the bundle of

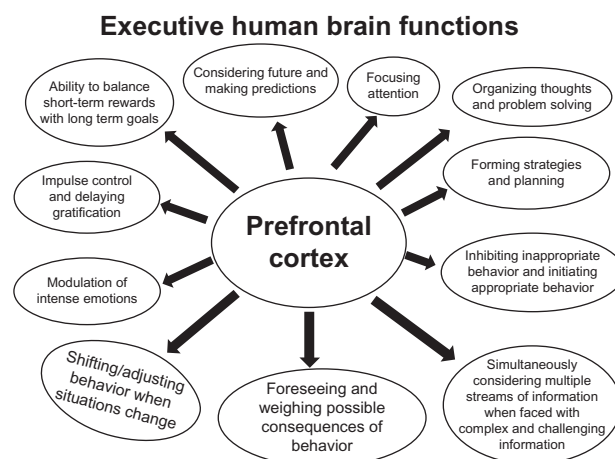


Figure 3 A diagram illustrating the developmental regulation of executive functions by the prefrontal cortex, which remains under construction during adolescence.

Notes: Several executive brain functions are governed by the prefrontal cortex, which remains in a state of active maturation during adolescence. These complex brain functions are regulated by the prefrontal cortex as illustrated in this figure (based on the original discoveries by Giedd and Steinberg).^{1,21–23,25} Due to immature functional areas in the prefrontal cortex, adolescent teens may take part in risk seeking behavior including unprotected sex, impaired driving, and drug addiction.

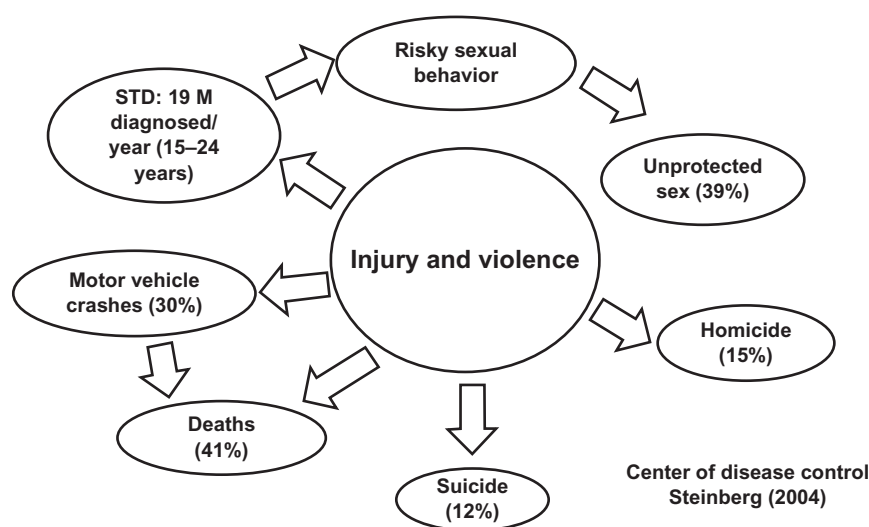


Figure 5 Leading cause of death among adolescents (10–24 years).

Notes: Injury and violence are the two most common leading causes of death during adolescence. Out of 19 million adolescents (15–24 years) in the US that were diagnosed with HIV/AIDS, 39% admitted that they had unprotected sex. In addition to risky sex behavior, 30% of adolescents had been involved in motor vehicle accidents, with 41% of these linked to deaths; 12% committed suicide; and 15% were victims of homicide as illustrated in this figure (Steinberg 2004, Centers for Disease Control and Prevention).¹⁸

Abbreviations: AIDS, acquired immune deficiency syndrome; HIV, human immunodeficiency virus; M, million; STD, sexually transmitted disease.

development correlate with the maturation of the frontal lobe, and is marked by a shift from the development of additional neural connections to synaptic pruning, as well as by an increase in the release of hormones, all of which drive an adolescent's mood and impulsive behavior.

By the age of 15 years, there is little difference in adolescents' and adults' decision-making patterns pertaining to hypothetical situations. Teens were found to be capable of reasoning about the possible harm or benefits of different courses of action; however, in the real world, teens still engaged in dangerous behaviors, despite understanding the risks involved.^{22,23,59} Hence, both the role of emotions and the connection between feeling and thinking need to be considered while studying the way teens make decisions.

Investigators have differentiated between “hot” cognition and “cold” cognition.²⁴ Hot cognition is described as thinking under conditions of high arousal and intense emotion. Under these conditions, teens tend to make poorer decisions. The opposite of hot cognition is cold cognition, which is critical and over-analyzing.²⁵ In cold cognition, circumstances are less intense and teens tend to make better decisions. Then, with the addition of complex feelings – such as fear of rejection, wanting to look cool, the excitement of risk, or anxiety of being caught – it is more difficult for teens to think through potential outcomes, understand the consequences of their decisions, or even use common sense.²⁶ The apparent immaturity of the connections between the limbic system,

prefrontal cortex, and the amygdala provides further support for this concept.

Sensation seeking

The nucleus accumbens, a part of the brain's reward system located within the limbic system, is the area that processes information related to motivation and reward. Brain imaging has shown that the nucleus accumbens is highly sensitive in adolescents, sending out impulses to act when faced with the opportunity to obtain something desirable.²⁷ For instance, adolescents are more vulnerable to nicotine, alcohol, and other drug addictions because the limbic brain regions that govern impulse and motivation are not yet fully developed.²⁸ During puberty, the increases in estrogen and testosterone bind receptors in the limbic system, which not only stimulates sex drive, but also increases adolescents' emotional volatility and impulsivity. Changes in the brain's reward sensitivity that occur during puberty have also been explored. These changes are related to decreases in DA, a neurotransmitter that produces feelings of pleasure.²⁹ Due to these changes, adolescents may require higher levels of DAergic stimulation to achieve the same levels of pleasure/reward, driving them to make riskier decisions.

Self-regulation

Self-regulation has been broadly classified as the management of emotions and motivation.³⁰ It also involves directing and controlling behavior in order to meet the challenges of

the environment and to work toward a conscious purpose. Self-regulation also entails controlling the expression of intense emotions, impulse control, and delayed gratification. As adolescents progress toward adulthood with a body that is almost mature, the self-regulatory parts of their brains are still maturing. An earlier onset of puberty increases the window of vulnerability for teens, making them more susceptible to taking risks that affect their health and development over a prolonged period.³¹

Behavioral control requires a great involvement of cognitive and executive functions. These functions are localized in the prefrontal cortex, which matures independent of puberty and continues to evolve up until 24 years of age. It has been suggested that, during this period, adolescents should not be overprotected, but be allowed to make mistakes, learn from their own experiences, and practice self-regulation. Parents and teachers can help adolescents through this period by listening and offering support and guidance.

Recently, Steinberg studied risk-taking behavior in teens and how this was influenced by their peers.³² He used a driving simulation game in which he studied teens deciding on whether or not to run a yellow light, and found that when teens were playing alone they made safer decisions, but in the presence of friends they made riskier decisions. When teens find themselves in emotionally arousing situations, with their immature prefrontal cortices, hot cognitive thinking comes into play, and these adolescents are more likely to take riskier actions and make impulsive decisions.

Societal influences

Mass media, community, and adult role models can also influence adolescent risk-taking behaviors. Teens are constantly exposed to emotionally arousing stimuli through multimedia, which encourages unprotected sex, substance abuse, alcohol abuse, and life-threatening activities.^{32,33} Even neighborhoods, friends, and communities provide teens with opportunities to engage in risky behaviors, although local law enforcement authorities regulate the purchase of cigarettes, access to and acceptability of guns, and the ability to drive cars. Even adults can have trouble resisting engaging in some of these risky behaviors; however, the temptation must be much harder for teens, whose judgment and decision-making skills are still developing.³⁴

Recent functional MRI studies have demonstrated the extent of development during adolescence in the white matter and grey matter regions within the social brain. Activity in some of these regions showed changes

between adolescence and adulthood during social cognition tasks. These studies have provided evidence that the concept of mind usage remains developing late in adolescence.^{1,21,33}

Substance abuse

The mechanisms underlying the long-term effects of prenatal substance abuse and its consequent elevated impulsivity during adolescence are poorly understood. Liu and Lester³⁴ have reported on developmentally-programmed neural maturation and highlighted adolescence as a critical period of brain maturation. These investigators have studied impairments in the DAergic system, the hypothalamic–pituitary–adrenal axis, and the pathological interactions between these two systems that originate from previous fetal programming in order to explain insufficient behavioral inhibition in affected adolescents. In addition, Burke³⁵ has examined the development of brain functions and the cognitive capabilities of teenagers. Specifically, these two sets of investigators have explored the effect of alcohol abuse on brain development, and the fundamental cognitive differences between adolescents and adults, and have suggested that the adultification of youth is harsh for those whose brains have not fully matured.

Cannabis

Cannabis is the most commonly consumed drug among adolescents, and its chronic use may affect maturational refinement by disrupting the regulatory role of the endocannabinoid system.³⁶ Adolescence represents a critical period for brain development and the endocannabinoid system plays a critical role in the regulation of neuronal refinement during this period. In animals, adolescent cannabinoid exposure caused long-term impairment in specific components of learning and memory, and differentially affected emotional reactivity with milder effects on anxiety behavior and more pronounced effects on depressive behavior.³⁷ Epidemiological studies have suggested that adolescent cannabis abuse may increase their risk of developing cognitive abnormalities, psychotic illness, mood disorders, and other illicit substance abuse later in life.^{36,38–40} Cannabis abuse in adolescence could increase the risk of developing psychiatric disorders, especially in people who are vulnerable to developing psychiatric syndromes. So far, only a few studies have investigated the neurobiological substrates of this vulnerability;⁵⁶ hence, further investigation is required to clarify the molecular mechanisms underlying the effect of cannabis on the adolescent brain.

Nicotine

Recent studies have provided a neural framework to explain the developmental differences that occur within the mesolimbic pathway based on the established role of DA in addiction.^{41,42} During adolescence, excitatory glutamatergic systems that facilitate DAergic neurotransmission are overdeveloped, whereas inhibitory GABAergic systems remain underdeveloped. DAergic pathways originate in the ventral tegmental area and terminate in the nucleus accumbens, where dopamine is increased by nicotine, but decreased during withdrawal. Thus, it has been hypothesized that adolescents display enhanced nicotine reward and reduced withdrawal via enhanced excitation and reduced inhibition of ventral tegmental area cell bodies that release DA in the nucleus accumbens.^{44,45} Although this framework focuses on both adolescents and adults, it may also apply to the enhanced vulnerability to nicotine in adults that were previously exposed to nicotine during adolescence, suggesting that the diagnostic criteria developed for nicotine dependence in adults (based primarily on withdrawal) may be inappropriate during adolescence, when nicotine withdrawal does not appear to play a major role in nicotine use.³⁹ Furthermore, treatment strategies involving nicotine replacement may be harmful for adolescents because it may cause enhanced vulnerability to nicotine dependence later in adulthood. Adolescents that initiate tobacco abuse are more vulnerable to long-term nicotine dependence. A unifying hypothesis has been proposed based on animal studies, and it suggests that adolescents (as compared to adults) experience enhanced short-term positive effects and reduced adverse effects toward nicotine, and they also experience fewer negative effects during nicotine withdrawal.³⁹ Thus, during adolescence, the strong positive effects associated with nicotine are inadequately balanced by the negative effects that contribute to nicotine dependence in adults.

Alcohol

Recently, the development of brain functions, the cognitive capabilities of adolescents, and the effect of alcohol abuse on brain maturation have been examined.^{49,50} Cognitive differences between adolescents and adults suggest that the adultification of youths is deleterious for youths whose brains have not fully matured. Adolescence is the time during which most individuals first experience alcohol exposure, and binge drinking is very common during this period.^{29,50,43} There is increasing evidence for long-lasting neurophysiological changes that may occur following exposure to ethanol during adolescence in animal models.⁵⁰ If alcohol exposure is

neurotoxic to the developing brain during adolescence, then understanding how ethanol affects the developing adolescent brain becomes a major public health issue. Adolescence is a critical time period when cognitive, emotional, and social maturation occurs and it is likely that ethanol exposure may affect these complex processes. During a period that corresponds to adolescence in rats, the relatively brief exposure to high levels of alcohol via ethanol vapors caused long-lasting changes in functional brain activity.⁵¹ The following observations were recorded: disturbances in waking electroencephalography; a reduction in the P3 wave (P3a and P3b) component of event-related potential measurements; reductions in the mean duration of slow-wave sleep; and the total amount of time spent in slow-wave sleep – findings that are consistent with the premature sleep patterns observed during aging.⁵⁰

Sex differences

Sex differences in many behaviors, including drug abuse, have been attributed to social and cultural factors.^{43,46} A narrowing gap in drug abuse between adolescent boys and girls supports this hypothesis;⁵² however, some sex differences in addiction vulnerability reflect biologic differences in the neurocircuits involved in addiction. A male predominance in overall drug abuse appears by the end of adolescence, while girls develop a rapid progression from the time of the first abuse to dependence, and this represents female-based vulnerability. Recent studies have emphasized the contribution of sex differences in the function of the ascending DAergic systems, which are critical in reinforcement.^{3,43} These studies highlight the behavioral, neurochemical, and anatomical changes that occur in the DAergic functions that are related to the addictions that occur during adolescence. In addition, these studies have presented novel findings about the emergence of sex differences in DAergic function during adolescence.^{43,46–48} Sex differences in drinking patterns and the rates of alcohol abuse and dependence begin to emerge during the transition from late puberty to young adulthood. Increases in pubertal hormones, including gonadal and stress hormones, are a prominent developmental feature of adolescence and could contribute to the progression of sex differences in alcohol drinking behavior during puberty. Witt⁴⁶ reviewed experimental and correlational studies of gonadal and stress-related hormone changes, as well as their effects on alcohol consumption and the associated neurobehavioral actions of alcohol on the mesolimbic dopaminergic system. Mechanisms have been suggested by which reproductive and stress-related hormones may modulate neural circuits within the brain reward system, and these hormones may produce sex differences in terms of

alcohol consumption patterns and adolescents' vulnerability to alcohol abuse and dependence, which become apparent during the late pubertal period.

Chemotherapy

Recently, Vázquez et al⁵³ emphasized the need for the early and accurate diagnosis of CNS complications during and after pediatric cancer treatment because of the improvement in overall survival rates related to innovative and aggressive oncologic therapies. A major concern in this issue is recognizing the radiologic features of these CNS complications. Radiologists are supposed to be familiar with the early and late effects of cancer therapy in the pediatric CNS (toxic effects, infection, endocrine or sensory dysfunction, neuropsychological impairment, and secondary malignancies) in order to provide an accurate diagnosis and to minimize morbidity. The acquisition of further knowledge about these complications will enable the development of more appropriate therapeutic decisions, effective patient surveillance, and an improved quality of life by decreasing the long-term consequences in survivors. Certain chemotherapeutic compounds and environmental agents, such as anesthetics, antiepileptics, sleep-inducing and anxiolytic compounds, nicotine, alcohol, and stress, as well as agents of infection have also been investigated quite extensively and have been shown to contribute to the etiopathogenesis of serious neuropsychiatric disorders.⁵⁴ All of these agents have a deleterious influence on developmental processes during the time when the brain experiences major changes in early childhood and during adulthood. Several of these agents have contributed to the structural and functional brain abnormalities that have been observed in the biomarker profiles of schizophrenia and fetal alcohol syndrome. The effects of these agents are generally permanent and irreversible.⁵⁴

Nutrition

The rapid expansion of knowledge in this field, from basic science to clinical and community-based research, is expected to lead to urgently needed research in support of effective, evidence-based medicine and treatment strategies for undernutrition, overnutrition, and eating disorders in early childhood. Eating is necessary for survival and provides a sense of pleasure, but may be perturbed, leading to undernutrition, overnutrition, and eating disorders. The development of feeding in humans relies on the complex interplay between homeostatic mechanisms; neural reward systems; and adolescents' motor, sensory, and emotional capabilities. Furthermore, parenting, social factors, and food influence the development of eating behavior.

Recently, the neural development of eating behavior in children has been investigated.⁵⁵ Furthermore, developmentally programmed neural maturation has been discussed in order to highlight adolescence as the second most critical period of brain maturation.⁵⁶ These studies used impairments of the DAergic system, the hypothalamic–pituitary–adrenal axis, and pathological interactions between these two systems originating from fetal programming in a dual-system model to explain insufficient behavioral inhibition in affected adolescents.

The range of exogenous agents, such as alcohol and cocaine, which are generally likely to detrimentally affect the development of the brain and CNS defies estimation, although the accumulated evidence is substantial.^{57–60} Pubertal age affects the fundamental property of nervous tissue excitability; excessive excitatory drive is seen in early puberty and a deficiency is seen in late puberty. It has been postulated that, with adequate fish oils and fatty acids, the risk of psychopathology can be minimized, whereas a deficiency could lead to subcortical dysfunction in early puberty, and a breakdown of cortical circuitry and cognitive dysfunctions in late puberty.⁶¹ Thus, postpubertal psychoses, schizophrenia, and manic–depressive psychosis during the pubertal age, along with excitability, may be the result of continuous dietary deficiency, which may inhibit the expression of the oligodendrocyte-related genes responsible for myelogenesis. The beneficial effect of fish oils and fatty acids in schizophrenia, fetal alcohol syndrome, developmental dyslexia, attention deficit hyperactivity disorder, and in other CNS disorders supports the hypothesis that the typical diet might be persistently deficient in the affected individuals, as illustrated in Figure 6. However, the amount of fish oils and fatty acids needed to secure normal brain development and function is not known. It seems conjectural to postulate that a dietary deficiency in fish oils and fatty acids is causing brain dysfunction and death; however, all of these observations tend to suggest that a diet focusing on mainly protein is deficient, and the deficiency is most pronounced in maternal nutrition and in infancy, which might have a deleterious impact on the maturation of the adolescent brain.

Conclusion

Neuromorphological, neurochemical, neurophysiological, neurobehavioral, and neuropharmacological evidence suggests that the brain remains in its active state of maturation during adolescence.^{1,7,19,21} Such evidence supports the hypothesis that the adolescent brain is structurally and functionally vulnerable to environmental stress, risky behavior, drug

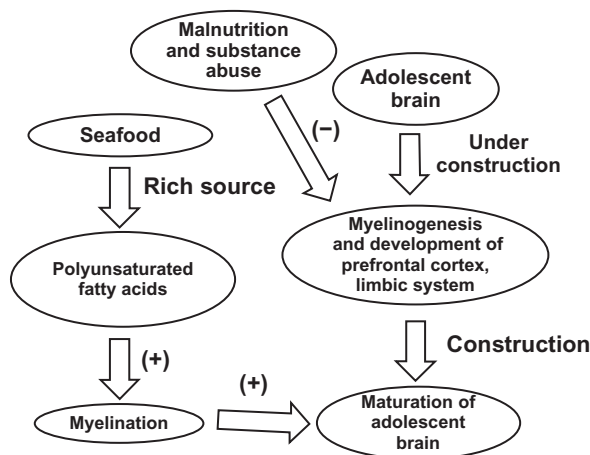


Figure 6 Effect of seafood on the maturation of the adolescent brain.

Notes: MRI studies have provided evidence that in addition to the prefrontal cortex and limbic system, myelinogenesis and neurocircuitry remains under construction during adolescence.^{1,7,19,21} Myelinogenesis requires precursors such as polyunsaturated fatty acids, of which many seafoods are a rich source. Hence, consuming seafood may accelerate brain maturation in adolescents. However, malnutrition and substance abuse may inhibit maturation of the adolescent brain. (+) induction; (–) inhibition.

addiction, impaired driving, and unprotected sex. Computed tomography and MRI studies also provide evidence in support of this hypothesis.¹⁹

Brain maturation occurs during adolescence due to a surge in the synthesis of sex hormones implicated in puberty including estrogen, progesterone, and testosterone. These sex hormones augment myelinogenesis and the development of the neurocircuitry involved in efficient neurocybernetics. Although tubulinogenesis, axonogenesis, and synaptogenesis can occur during the prenatal and early postnatal periods, myelinogenesis involved in the insulation of axons remains under construction in adolescence. Sex hormones also significantly influence food intake and sleep requirements during puberty. In addition to dramatic changes in secondary sex characteristics, sex hormones may also influence the learning, intelligence, memory, and behavior of adolescents.

Furthermore, it can be observed that the development of excitatory glutamatergic neurotransmission occurs earlier in the developing brain as compared to GABAergic neurotransmission, which makes the pediatric population susceptible to seizures.

The development and maturation of the prefrontal cortex occurs primarily during adolescence and is fully accomplished at the age of 25 years. The development of the prefrontal cortex is very important for complex behavioral performance, as this region of the brain helps accomplish executive brain functions.

A detailed study is required in order to determine the exact biomarkers involved, as well as the intricate influence of

diet, drugs, sex, and sleep on the maturation of the adolescent brain as discussed briefly in this report.

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References

- Giedd JN, Blumenthal J, Jeffries NO, et al. Brain development during childhood and adolescence: a longitudinal MRI study. *Nat Neurosci.* 1999;2(10):861–863.
- Li K, Xu E. The role and the mechanism of gamma-aminobutyric acid during central nervous system development. *Neurosci Bull.* 2008;24(3):195–200.
- Wahlstrom D, Collins P, White T, Luciana M. Developmental changes in dopamine neurotransmission in adolescence: behavioral implications and issues in assessment. *Brain Cogn.* 2010;72(1):146–159.
- Kaplan PS. *Adolescence*. Boston, MA: Houghton Mifflin Company; 2004.
- Gavin L, MacKay AP, Brown K, et al; Centers for Disease Control and Prevention (CDC). Sexual and reproductive health of persons aged 10–24 years – United States, 2002–2007. *MMWR Surveill Summ.* 2009;58(6):1–58.
- Sylvester R. *The Adolescent Brain: Reaching for Autonomy*. Newbury Park (CA): Corwin Press; 2007.
- Baird AA, Gruber SA, Fein DA, et al. Functional magnetic resonance imaging of facial affect recognition in children and adolescents. *J Am Acad Child Adolesc Psychiatry.* 1999;38(2):195–199.
- Frontline: Inside the Teenage Brain [webpage on the Internet]. Arlington (TX): Public Broadcasting Service; 2002. Available from: <http://www.pbs.org/wgbh/pages/frontline/shows/teenbrain/>. Accessed August 6, 2009.
- Dahl RE. Beyond raging hormones: the tinderbox in the teenage brain. *Cerebrum.* 2003;5(3):7–22.
- Blakemore SJ. Development of the social brain in adolescence. *J R Soc Med.* 2012;105(3):111–116.
- Sisk CL, Foster DL. The neural basis of puberty and adolescence. *Nat Neurosci.* 2004;7(10):1040–1047.
- Peper JS, van den Heuvel MP, Mandl RC, Hulshoff Pol HE, van Honk J. Sex steroids and connectivity in the human brain: a review of neuroimaging studies. *Psychoneuroendocrinology.* 2011;36(8):1101–1113.
- Choudhury S, Blakemore SJ, and Charman T. Social cognitive development during adolescence. *Soc Cogn Affect Neurosci.* 2006;1(3):165–174.
- den Bos (2011) W.V *The neurocognitive development of social decision making*. Doctoral Research Thesis. (P 1–189) Amsterdam.
- Somerville LH, Fani N, and Erin B. McClure-Tone E.B. Behavioral and neural representation of emotional facial expressions across the lifespan. *Dev Neuropsychol.* 2011;36(4):408–428.
- Sales JM and Irvin CE. Theories of adolescent risk taking (2009) The biopsychological model. In *Adolescent Health*. Diclemente R.J, Santelli, J.S, Crosby RA (Eds) (pp 31–50) San Francisco: John Wiley and Sons.
- Frontline: Interview Deborah Yurgelun-Todd [webpage on the Internet]. Arlington: Public Broadcasting Service; 2002. Available from: <http://www.pbs.org/wgbh/pages/frontline/shows/teenbrain/interviews/todd.html>. Accessed February 14, 2013.
- Guy AE, McClure-Tone EB, Shiffrin ND, Pine DS, Nelson EE. Probing the neural correlates of anticipated peer evaluation in adolescence. *Child Dev.* 2009;80(4):1000–1015.

19. Casey BJ, Jones RM, Hare TA. The adolescent brain. *Ann NY Acad Sci.* 2008;1124:111–126.
20. Walsh D, Bennett N. *Why Do They Act That Way? A Survival Guide to the Adolescent Brain for You and Your Teen.* New York: Simon and Schuster; 2004.
21. Giedd JN. Structural magnetic resonance imaging of the adolescent brain. *Ann NY Acad Sci.* 2004;1021:77–85.
22. Steinberg L. Risk taking in adolescence: what changes and why? *Ann NY Acad Sci.* 2004;1021:51–58.
23. Steinberg L. Cognitive and affective development in adolescence. *Trends Cogn Sci.* 2005;9(2):69–76.
24. Abelson, R. P. (1963). Computer simulation of “hot cognition”, in S. S. Tomkins and S. Messick (Eds.), *Computer simulation of personality* (pp. 277–302). New York: Wiley.
25. Ziva K (1990). “The case for motivated reasoning”. *Psychological Bulletin* 108(3): 480–498.
26. Benes FM. The development of the human frontal cortex: The maturation of neurotransmitter system and their interactions. In: Nelson CA, Luciana M, editors. *Handbook of Developmental Cognitive Neuroscience.* Cambridge, MA: MIT Press; 2001:79–92.
27. Gardner M, Steinberg L. Peer Influence on risk taking, risk preference and risky decision-making in adolescence and adulthood. *Dev Psychol.* 2005;41(4):625–635.
28. <http://www.hhs.gov> [homepage on the Internet]. New Research on Adolescent Brain Development. Center for Substance Abuse Prevention; 2004. http://www.hhs.gov/opa/familylife/tech_assistance/etraining/adolescent_brain/risk_taking/changes/sensation_seeking/index.html#fn3. Accessed March 14, 2013.
29. Lopez B, Schwartz SJ, Prado G, Campo AE, Pantin H. Adolescent neurological development and implications for adolescent substance abuse prevention. *J Prim Prev.* 2008;29(1):5–35.
30. Steinberg L, Belsky J. An evolutionary perspective on psychopathology in adolescence. In: Cicchetti D, Toth SL, editors. *Adolescence: Opportunities and Challenges: Volume 7 of Rochester Symposium on Developmental Psychology Series.* Rochester, NY: University of Rochester Press; 1996:93–124.
31. Simpson RA. Raising Teens: A Synthesis of Research and a Foundation for Action. Center for Health Communication, Harvard School of Public Health. 2001. Available from: <http://www.hsph.harvard.edu/chc/parenting/report.pdf>.
32. Steinberg L. A social neuroscience perspective on adolescent risk-taking. *Dev Rev.* 2008;28(1):78–106.
33. Blakemore SJ. Development of the social brain in adolescence. *J R Soc Med.* 2012;105(3):111–116.
34. Liu J, Lester BM. Reconceptualizing in a dual-system model the effects of prenatal cocaine exposure on adolescent development: a short review. *Int J Dev Neurosci.* 2011;29(8):803–809.
35. Burke AS. Under construction: brain formation, culpability, and the criminal justice system. *Int J Law Psychiatry.* 2011;34(6):381–385.
36. Palmer RH, Young SE, Hopfer CJ, et al. Developmental epidemiology of drug use and abuse in adolescence and young adulthood: evidence of generalized risk. *Drug Alcohol Depend.* 2009;102(1–3):78–87.
37. Bossong NG, Niesink RJ. Adolescent brain maturation, the endogenous cannabinoid system and the neurobiology of cannabis-induced schizophrenia. *Prog Neurobiol.* 2010 Nov;92(3):370–385.
38. Vik P, Brown SA. Life events and substance abuse during adolescence. In: Miller TW, editor. *Children of Trauma.* Madison, CT: International Universities Press; 1998:179–204.
39. Rubino T, Zamberletti E, Parolaro D. Adolescent exposure to cannabis as a risk factor for psychiatric disorders. *J Psychopharmacol.* 2012;26(1):177–188.
40. Gonzalez R, Swanson JM. Long-term effects of adolescent-onset and persistent use of cannabis. *Proc Natl Acad Sci U S A.* 2012;109(40):15970–15971.
41. O'Dell LE. A psychobiological framework of the substrates that mediate nicotine use during adolescence. *Neuropharmacology.* 2009;56 Suppl 1: 263–278.
42. Philpot R, Kirstein C. Developmental Differences in the Accumbal Dopaminergic Response to Repeated Ethanol Exposure. *Ann. NY Acad. Sci.* 2004;1021:422–426.
43. Kuhn C, Johnson M, Thomae A, et al. The emergence of gonadal hormone influences on dopaminergic function during puberty. *Horm Behav.* 2010;58(1):122–137.
44. Burke AS. Under construction: brain formation, culpability, and the criminal justice system. *Int J Law Psychiatry.* 2011;34(6):381–385.
45. Spear LP. Adolescent period: biological basis of vulnerability to develop alcoholism and other ethanol-mediated behaviors. In: Noronha A, Eckardt M, Warren K, editors. *Review of NiAAA's Neuroscience and Behavioral Research Portfolio.* Bethesda, MD: National Institute on Alcohol Abuse and Alcoholism; 2000:315–333.
46. Witt ED. Puberty, hormones, and sex differences in alcohol abuse and dependence. *Neurotoxicol Teratol.* 2007;29(1):81–95.
47. Nolen-Hoeksema S, Girgus JS. The emergence of gender differences in depression during adolescence. *Psychological Bulletin.* 1994;115(3):424–443.
48. Lenroot RK, Giedd JN. Sex differences in the adolescent brain. *Brain Cogn.* 2010;72(1):1–19.
49. Spear LP. The adolescent brain and age-related behavioral manifestations. *Neurosci Biobehav Rev.* 2000;24(4):417–463.
50. Ehlers CL, Criado JR. Adolescent ethanol exposure: does it produce long-lasting electrophysiological effects? *Alcohol.* 2010;44(1):27–37.
51. Allen CD, Lee S, Koob GF, Rivier C. Immediate and prolonged effects of alcohol exposure on the activity of the hypothalamic-pituitary-adrenal axis in adult and adolescent rats. *Brain Behav Immun.* 2011 June;25(Suppl 1):S50–S60.
52. Schulte MT, Ramo D, and Brown SA. Gender Differences in Factors Influencing Alcohol Use and Drinking Progression Among Adolescents. *Clin Psychol Rev.* 2009 August;29(6):535–547.
53. Vázquez E, Delgado I, Sánchez-Montañez A, Barber I, Sánchez-Toledo J, Enríquez G. Side effects of oncologic therapies in the pediatric central nervous system: update on neuroimaging findings. *Radiographics.* 2011;31(4):1123–1139.
54. Archer T. Effects of exogenous agents on brain development: stress, abuse and therapeutic compounds. *CNS Neurosci Ther.* 2011;17(5):470–489.
55. Gahagan S. Development of eating behavior: biology and context. *J Dev Behav Pediatr.* 2012;33(3):261–271.
56. Sisk CL and Foster DL. The neural basis of puberty and adolescence. *Nature Neuroscience* 2004;7:1040–1047.
57. Liu J, Lester BM. Reconceptualizing in a dual-system model the effects of prenatal cocaine exposure on adolescent development: a short review. *Int J Dev Neurosci.* 2011;29(8):803–809.
58. Saugstad LF. From superior adaptation and function to brain dysfunction—the neglect of epigenetic factors. *Nutr Health.* 2004;18(1):3–27.
59. Steinberg L. Risk taking in adolescence: new perspectives from brain and behavioral science. *Curr Dir Psychol Sci.* 2007;16(2):55–59.
60. Brown SA, Tapert SF, Granholm E, Delis DC. Neurocognitive functioning of adolescents: effects of protracted alcohol use. *Alcohol Clin Exp Res.* 2000;24(2):164–171.
61. Rayyan M, Devlieger H, Jochum F, Allegaert K. Short-Term Use of Parenteral Nutrition With a Lipid Emulsion Containing a Mixture of Soybean Oil, Olive Oil, Medium-Chain Triglycerides, and Fish Oil. A Randomized Double-Blind Study in Preterm Infants. *JPEN J Parenter Enteral Nutr.* 2012 January;36(1 suppl):81S–94S.

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EXHIBIT 2



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Review Article

A time of change: Behavioral and neural correlates of adolescent sensitivity to appetitive and aversive environmental cues

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ABSTRACT

Adolescence is a developmental period that entails substantial changes in affective and incentive-seeking behavior relative to both childhood and adulthood, including a heightened propensity to engage in risky behaviors and experience persistent negative and labile mood states. This review discusses the emotional and incentive-driven behavioral changes in adolescents and their associated neural mechanisms, focusing on the dynamic interactions between the amygdala, ventral striatum, and prefrontal cortex. Common behavioral changes during adolescence may be associated with a heightened responsiveness to incentives and emotional cues while the capacity to effectively engage in cognitive and emotion regulation is still relatively immature. We highlight empirical work in humans and animals that addresses the interactions between these neural systems in adolescents relative to children and adults, and propose a neurobiological model that may account for the nonlinear changes in adolescent behavior. Finally, we discuss other influences that may contribute to exaggerated reward and emotion processing associated with adolescence, including hormonal fluctuations and the role of the social environment.

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1. Introduction

The description of adolescence as “a developmental period rife with change” may be an understatement for those of us who think back to our own experiences during this time of life, or who observe teens today (Hall, 1904). Adolescence can be defined as the phase of gradual transition between childhood and adulthood, which is overlapping yet conceptually distinct from the physical changes marking puberty and physical maturation (Ernst, Pine, & Hardin, 2006; Spear, 2000). In recent years, researchers from a broad spectrum of scientific disciplines have shown significant interest in this period of the lifespan due to its intense physical, behavioral, social, and neurological changes, and the alarming health statistics associated with this time of life.

Beyond the intellectual interest in this period as a psychological snapshot in time, research examining adolescent behavior and its associated neural changes is particularly relevant to adolescent health. In adolescence, there is a heightened propensity to engage in risky behaviors that can lead to negative outcomes, including

substance abuse, unprotected sex, inflicting harm on others, injuries, and death. According to the 2007 Youth Risk Behavior Survey (YRBS, Eaton et al., 2008) the four leading causes of death that account for 72% of adolescent mortality – motor vehicle accidents, unintentional injuries, homicide, and suicide – are preventable. Such statistics suggest that these fatalities may be attributed, in part, to poor choices or risky actions (e.g., accidents, injuries) and/or heightened emotionality (e.g., suicide) underscoring the importance of understanding the biological basis of emotional and incentive-seeking behavior of adolescents, the focus of the present review.

2. Storm and stress? Affective changes during adolescence

Adolescence has been considered, almost by definition, as a period of heightened stress (Spear, 2000) due to the array of transitions being experienced concomitantly, including physical maturation, drive for independence, increased salience of social and peer interaction, and brain development (Blakemore, 2008; Casey, Getz, & Galvan, 2008; Casey, Jones, & Hare, 2008). Although new-found independence and social engagement can be stimulating and challenging in a positive way, it may also lead to feelings of being overwhelmed by change, which has historically led some researchers to

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characterize adolescence as ridden with 'storm and stress' (Hall, 1904). The controversial 'storm and stress' viewpoint is bolstered by reports that the onset of many psychiatric illnesses increases sharply from childhood to adolescence (Compas, Orosan, & Grant, 1993), with the lifetime risk for the emergence of mental illness peaking at 14 years of age (Kessler et al., 2005). Although a full discussion of clinical adolescent populations is of inherent interest to this topic, it is outside the scope of the present review and we refer the reader to existing articles that address these issues in greater detail (Paus, Keshavan, & Giedd, 2008; Steinberg, 2005).

In terms of the typical range of emotions, certain classes of emotional states – particularly negative emotional states – show a peak in prevalence during adolescence (Compas, Hinden, & Gerhardt, 1995; Petersen et al., 1993; Rutter, Graham, Chadwick, & Yule, 1976). Most recently, YRBS results showed that in the prior year, more than one in four adolescents (27.3%) had experienced significant symptoms of depression for at least two weeks, to the point that it interfered with their everyday functioning (Eaton et al., 2008). Experiencing frequent negative affect is particularly common during the early adolescent years, more so in females than males (Larson, Moneta, Richards, & Wilson, 2002), and in addition to sad mood, also manifests itself in anxiety (Abe & Suzuki, 1986), self-consciousness, and low self-esteem (Simmons, Rosenberg, & Rosenberg, 1973; Thornburg & Jones, 1982). Feeling sad, depressed, or hopeless may be associated with the heightened rates of affective disorders, attempted and completed suicide, and addiction also observed during adolescence (Mościcki, 2001; Pine, Cohen, & Brook, 2001; Silveri, Tzilos, Pimentel, & Yurgelun-Todd, 2004; Steinberg, 2005). These statistics underscore the need to understand the physiological basis of these emotional state changes in adolescents.

Finally, adolescents' negative emotional states are not only frequent but their emotional responses also tend to be more intense, variable and subject to extremes relative to adults (Arnett, 1999; Buchanan, Eccles, & Becker, 1992; Eccles et al., 1989; Simmons & Blyth, 1987). Larson and colleagues (2002) performed a cross-sectional beeper study that sampled the momentary affect experienced by early adolescents several times per day for a week, and then retested those individuals approximately 3 years later, after they had transitioned into late adolescence. Results indicated that early adolescents, defined here as fifth to eighth graders, experienced substantially greater short-term variability in affective state relative to what the same individuals experienced in ninth to twelfth grades (Larson et al., 2002). This study and others suggest that adolescent emotional states tend to be more labile than children and adults, and this appears to be particularly true during the early adolescent years.

The work just described paints a relatively bleak picture, suggesting that adolescence is doomed to be a very negative time of life. However, it is important to note that most adolescents are actually not miserable, and negotiate this potentially difficult period with relative ease and without lasting problems (Steinberg, 2008). We believe that a bias in available data may contribute to this discrepancy – while many studies ask adolescents to report on their negative emotions, very few ask about positive emotions which may also be elevated during this time (see Ernst et al., 2005). Consequentially, a more current view of adolescent affect is not deterministic with regard to experiencing 'storm and stress', but contends that being an adolescent may be a risk factor for experiencing intense negative emotional states (Arnett, 1999).

3. Adolescent incentive-driven behavior

In the previous section, we have asserted that adolescents frequently experience negative and volatile emotions. However, the

period of adolescence is also marked by a nonlinear enhancement in risk-taking behavior, characterized by approaching pleasurable experiences without appropriate reverence to their associated potentially negative consequences. Several classes of epidemiological data support this conceptualization of adolescent behavior. In particular, adolescents engage in significantly more risky driving, illicit drug use, criminal acts and unsafe sexual behavior than children and adults (Eaton et al., 2008; National Research Council, 2007; Substance Abuse and Mental Health Services Administration, 2007). These health statistics suggest that adolescents are risk-takers, but environmental influences such as reduced parental supervision and increased access to risk-enabling situations could also explain the increase in risk-taking between childhood and adolescence.

Empirical work measuring risk-taking in controlled environments has largely supported the notion that adolescents show disproportionate risk-taking in the absence of differential environmental demands. Cauffman and colleagues (in press) used the Iowa Gambling Task to test participants varying in age from pre-adolescence (10 years old) to adulthood (up to 30 years old). Using this task, approach- and avoidance-based decision-making was calculated separately by quantifying participants' ability to use experimenter feedback to learn to approach 'good' decks of cards (positive feedback) and avoid 'bad' decks (negative feedback). They found that levels of approach toward potential reward took on a curvilinear function, with the maximal sensitivity to positive feedback occurring during the adolescent years. In contrast, use of negative feedback to avoid negative outcomes strengthened with age in a linear fashion, not showing full maturity until the adult years. These findings suggest that adolescents may have a disproportionate approach orientation, paired with an immature avoidance orientation, which may explain the nonlinear boost in risk-taking behavior. These findings are consistent with the results of Figner, Mackinlay, Wilkening, and Weber (2009a), who employed the Columbia Card Task, a risky decision-making task with 'hot', or affectively-driven, and 'cold', deliberative decision making contexts. They observed that in the 'hot' condition, adolescents showed an increase in risk-taking relative to adults. Recently, this sample has been extended to individuals as young as 10 years of age, with findings indicating that pre-adolescents display a level of risk-taking comparable to adults, and less than adolescents (Figner, Mackinlay, Wilkening, & Weber, 2009b). These experiments lend support to the notion that adolescents are disproportionately motivated to approach potential rewards, particularly in contexts with heightened arousal or salience.

Why do adolescents display greater propensity for risk taking? Although the answer is complex and addressed by another article in this volume (see article by Doremus-Fitzwater, Verlinskaya, & Spear), risky behaviors observed in adolescence are likely related to an enhanced motivation to seek out incentives and new experiences. This drive may be mediated by a greater salience of rewarding stimuli during this age relative to children or adults (Steinberg, 2008) – in other words, a sensitization to reward (Casey, Getz, & Galvan, 2008; Casey, Jones, & Hare, 2008; Fareri, Martin, & Delgado, 2008). This interpretation is consistent with the behavioral findings just described, a documented enhancement of sensation seeking in adolescents relative to children and adults (Zuckerman, Eysenck, & Eysenck, 1978), enhanced reported positive affect following the receipt of a monetary reward (Ernst et al., 2005), and neurobiological evidence which will be discussed in the forthcoming sections. Interestingly, rodents also show enhanced novelty and sensation seeking during adolescence, suggesting that reward-seeking behavior is governed by primitive biological mechanisms (Adriani, Chiarotti, & Laviola, 1998; Laviola, Macri, Morley-Fletcher, & Adriani, 2003).

In humans, this tendency paired with an immature "self-regulatory competence" leads to heightened risk for poor choice behavior

(Steinberg, 2004). When placed in an emotionally salient situation, enhanced sensitivity to positive environmental cues biases adolescent behavior toward approaching incentives, even when that choice may be suboptimal or risky (Casey, Getz, et al., 2008; Casey, Jones, et al., 2008). Importantly, risky behavior cannot be explained by a deficiency in comprehending the potential consequences of these actions (Reyna & Farley, 2006). Adolescents are cognitively able to appreciate the objective riskiness of their behaviors, yet in the moment these warnings are not heeded, perhaps due to a variety of influences including peers, environmental context, or internal emotional state (Gardener & Steinberg, 2005; Steinberg, 2005), leading environmental cues to ‘win’ over cognitive control in emotionally charged circumstances. This conceptualization proposes that disproportionate sensitivity to salient environmental cues can partially account for the nonlinear increase in risky reward-seeking behavior during this stage of development.

Although at first glance, risky adolescent behavior may appear inconsistent with adolescents’ frequent experience of negative mood states, these tendencies need not be mutually exclusive (Bogin, 1994; Spear, 2000). Indeed, negative and extreme emotional behavior paired with increased risk-taking may facilitate evolutionarily appropriate behavior (Casey, Getz, et al., 2008; Casey, Jones, et al., 2008; Spear, 2000). Risk-taking and novelty seeking can be viewed as facilitatory to some of the primary goals of adolescence in societal structures in which individuals must leave their home territory – “testing out” one’s independence, generating sufficient motivation to explore new environments, and developing bonds with non-family members (including potential mates). A propensity to generate reactive and extreme emotions may complement this process of striving for independence. Labile and negative emotions may signal a heightened state of vigilance toward threat and safety cues, which may serve a greater importance when engaging in risk. As such, the combination of emotionality and incentive seeking may have come about for good reason, but in present society serves less of an adaptive purpose.

4. Synthesizing a model of adolescent behavior change

Based on the behavioral work just described, we have observed three main themes characterizing unique aspects of adolescent behavior, relative to behavior of children and adults. First, adolescents appear to show heightened sensitivity to salient environmental cues. Behaviorally, this idea is supported by epidemiological reports of adolescent risk-taking behavior, and empirical work showing exaggerated responses to both positive and negative environmental cues in adolescents relative to children and adults. What may seem like a mildly annoying or hurtful event to adults may constitute an intense emotional trigger in adolescents leading to strong negative affect. Similarly, an environmental cue signaling a potential source of hedonic pleasure may drive incentive-seeking behavior to a greater extent than in children or adults due to a heightened sensitivity to potential rewards.

A second theme in the characterization of adolescent behavior is that adolescents are often unable to exert behavioral control in the face of environmentally salient cues, leading to risky and potentially dangerous choice behaviors. In particular, adolescents are able to comprehend and reason the outcomes of suboptimal decisions. Yet, in the right context, be it with peers or in a certain mood state, adolescents approach salient environmental cues even when it is disadvantageous or potentially dangerous. In terms of controlling negative affect, a lack of prefrontal control may lead to deficient emotional regulation abilities, resulting in affective responses left ‘unchecked’ and resulting in highly emotional output.

Lastly, although adolescents tend to show heightened affective responsiveness and incentive-based behavior changes, these re-

sponses are highly subject to individual differences. It is easy to forget that many adolescents make rational decisions, and have no problem regulating their emotions. However, we believe that adolescence is a time of life that is, consistent with more current views on ‘storm and stress’ (Arnett, 1999), a risk factor for heightened emotionality. This stage of life, combined with predisposing factors such as individual differences in trait anxiety or mood, or state contextual factors such as the stability of family or peer relations, may constitute a compounded source of risk for experiencing intense emotional states observed during adolescence.

5. Toward a neurobiological model of adolescent behavior

We have developed a biological model that characterizes brain changes underlying the patterns of adolescent behavior that takes into account the nonlinearity of emotional and incentive-seeking behaviors that are unique to this period (Casey, Getz, et al., 2008; Casey, Jones, et al., 2008). This empirically driven model posits an imbalance between the relative structural and functional maturity of brain systems critical to emotional and incentive-based behavior (e.g., subcortical regions including the amygdala and ventral striatum) as compared to brain systems mediating cognitive and impulse control (e.g., the prefrontal cortex), see Fig. 1. A relative maturity of subcortical structures compared to a still immature prefrontal control system may enable strong signaling of subcortical systems paired with weak control signaling, to account for the biased emotional and incentive-based behavior that is typical of adolescence. This is in contrast with the periods of childhood, when both brain systems are relatively immature, and adulthood, when both brain systems are relatively mature – and in both cases, more balanced in their influence over behavior. The following section will discuss empirical research outlining the development, structure, and function of subcortical and prefrontal control brain systems and their interaction, as well as how imbalanced engagement of these systems can lead to the emotional and reward-seeking behaviors associated with adolescence.

We will focus primarily on three interacting brain systems whose dynamic functions are critical to adolescent emotional, incentive, and cognitive control behaviors. The amygdaloid complex, a cluster of nuclei situated in the medial temporal lobe, plays a critical role in processing information of biological significance (Aggleton, 2000; Davis & Whalen, 2001; LeDoux, 2000), including emotionally evocative stimuli, potential threats, and cues depicting the emotional states of others. A second critical player in this circuitry is the ventral striatum, a portion of the basal ganglia that

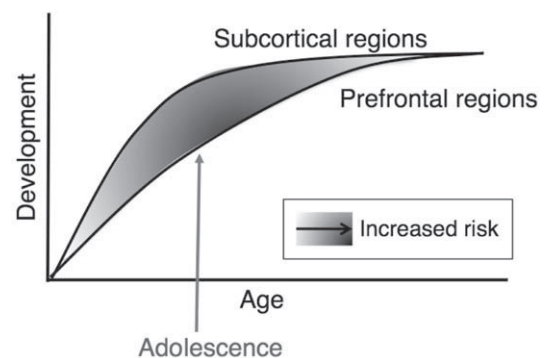


Fig. 1. Model for enhanced affective and incentive-based behavior in adolescence. Early maturation of subcortical regions such as the amygdala and ventral striatum (top line), combined with late maturation of prefrontal cortical regions (bottom line), predicts a nonlinear enhancement in affectively-driven behavior during adolescence.

contains the nucleus accumbens (NAcc). The NAcc contributes to decision-making behavior by signaling the anticipation and attainment of rewards, and serves to influence motivated behavior via connections with the prefrontal cortex (Cardinal, Parkinson, Hall, & Everitt, 2002; Delgado, 2007; Schultz, 2006). Finally, the prefrontal cortex has been implicated in wide-serving cognitive functions including the implementation of cognitive control, regulation of emotion, rational decision-making and complex cognition (Casey, Galvan, & Hare, 2005; Miller & Cohen, 2001; Ochsner & Gross, 2005). It is an imbalance between the relative maturity of the amygdala and NAcc, relative to the PFC, that we believe gives rise to the tendency toward disproportionate emotional and reward-sensitive behavior in adolescence.

6. Assessing differential relative maturity of subcortical and prefrontal regions

Outside of the functional neuroimaging literature, there is evidence to suggest a differential relative maturity of subcortical brain structures as compared to prefrontal regions, which may be most pronounced during adolescence. Evidence for the continued pruning of prefrontal cortical synapses well into development has been established in both nonhuman primates and humans (Huttenlocher, 1997; Rakic, Bourgeois, Eckenhoff, Zecevic, & Goldman-Rakic, 1986), with greater regional differentiation found in the human brain (Huttenlocher, 1997) such that cortical sensory and subcortical areas undergo dynamic synaptic pruning earlier than higher-order association areas. This conceptualization of cortical development is consistent with anatomical MRI work demonstrating protracted pruning of gray matter in higher-order prefrontal areas that continues through adolescence (e.g., Giedd et al., 1999) relative to subcortical regions. The amygdala and nucleus accumbens also show anatomical changes during this time of life but to a lesser degree. In an anatomical MRI experiment, gray matter measurements of the nucleus accumbens were not predicted by age, unlike prefrontal regions that were strongly negatively predicted by age (Sowell, Trauner, Gamst, & Jernigan, 2002). In terms of amygdala maturation, volumetric analyses of the human amygdala showed a substantially reduced slope of change magnitude relative to cortical areas in 4–18 year olds (Giedd et al., 1996). Taken together, these findings suggest a protracted developmental timecourse of the prefrontal cortex relative to these subcortical regions.

Our model is similar to other models of adolescent brain development (Nelson, Leibenluft, McClure, & Pine, 2005; Steinberg, 2008). However, the present model differs in that it attempts to account for adolescent changes in the processing of both appetitive and aversive cues, and emphasizes the dynamic interplay between subcortical and cortical brain systems across development. Finally, the current model integrates findings from children, adolescents and adults in order to account for the nonlinear nature of adolescent behavior change, and incorporates the important role of individual differences in modulating behavioral and brain responsivity.

7. Brain mechanisms of enhanced sensitivity to salient environmental cues

Functional neuroimaging techniques allow for the noninvasive measurement of regional brain activity while subjects perform tasks aimed at isolating psychological processes of interest. In affective neuroscience, researchers have used neuroimaging techniques to identify a network of brain regions that appear to be particularly responsive to appetitive and aversive stimuli, including the amygdala, ventral striatum, midbrain nuclei, and medial and lateral prefrontal cortices (Adolphs, 2002; Kober et al., 2008).

One can then look across a developmental trajectory to determine how the recruitment of emotion- and incentive-sensitive brain regions changes as a function of development, behavior, and individual differences.

Several neuroimaging experiments have examined the nature of subcortical responsivity to aversive and appetitive environmental cues during adolescence. Early work on this topic documented that adolescents showed a reliable amygdala response to facial expressions of emotion, including fearful faces (Baird et al., 1999). Subsequent experiments including an adult comparison group reported that adolescents elicited a greater amygdala response magnitude to negatively valenced facial expressions relative to adults (Guye, Monk, et al., 2008; Monk et al., 2003). However, it should be noted that this effect has not always been observed, as Thomas et al. (2001) documented an increase in amygdala response to neutral relative to fearful facial expressions in a pre-adolescent sample, the opposite effect of what was observed in adults. In addition, there is some evidence that the amygdala response in adolescents may be valence-independent, as adolescents also show enhanced amygdala activity to happy relative to neutral facial expressions (Williams et al., 2006), consistent with what is observed in adults (Somerville, Kim, Johnstone, Alexander, & Whalen, 2004).

Most recently, research has focused on tracking changes in neural responses to emotional cues during the transition into, during, and out of adolescence (Casey, Tottenham, Liston, & Durston, 2005) in order to detect nonlinear effects during this period of life. By testing individuals ranging in age from middle childhood to adulthood, it was observed that the response magnitude of the amygdala was significantly larger in adolescents compared to both children and adults, who showed comparable amygdala recruitment in response to facial expressions of emotion (Hare et al., 2008, see Fig. 2A). These studies and others have led to the interim conclusion that adolescents show an exaggeration in amygdala responsivity to emotional facial expressions relative to children and adults (Somerville, Fani, & McClure-Tone, in press). However, these patterns are not thought to be specific to facial expressions, as other negative cues such as the omission of a large monetary reward has been shown to generate disproportionately large amygdala responses in adolescents relative to adults as well (Ernst et al., 2005).

Functional neuroimaging techniques also have examined the neural underpinnings of adolescents' enhanced sensitivity to appetitive cues by using variations on incentive-related decision tasks, where subjects' behavioral choices determined the win or loss of money and/or magnitude of reward. These experiments have focused on the activity of the ventral striatum, which is sensitive to reward anticipation and learning in both the human (Delgado, Nystrom, Fissell, Noll, & Fiez, 2000; Knutson, Adams, Fong, & Hommer, 2001; O'Doherty, Deichmann, Critchley, & Dolan, 2002) and animal (Schultz, Dayan, & Montague, 1997). May and colleagues (2004) tested adolescent participants during a gambling task in which they could win or lose money on each trial, probing neural activity to the processing of reward outcomes. When comparing win to loss trials, adolescent participants recruited similar brain regions to what had been shown previously using the same task in adults (Delgado et al., 2000), including heightened activity in the ventral striatum. Interestingly, the ventral striatal timecourse of the reward response was temporally extended in adolescents compared to adults (Fareri et al., 2008), suggesting a temporal exaggeration in striatal recruitment to rewards. Using another gambling task, Ernst and colleagues (2005) measured neural activity and subjective affective responses to the wins and losses during fMRI scanning. Relative to adults, adolescents reported an exaggeration in subjective happiness experienced when winning large rewards, and these large reward trials elicited exaggerated neural responses within the NAcc. Taken together, these two experiments lend sup-

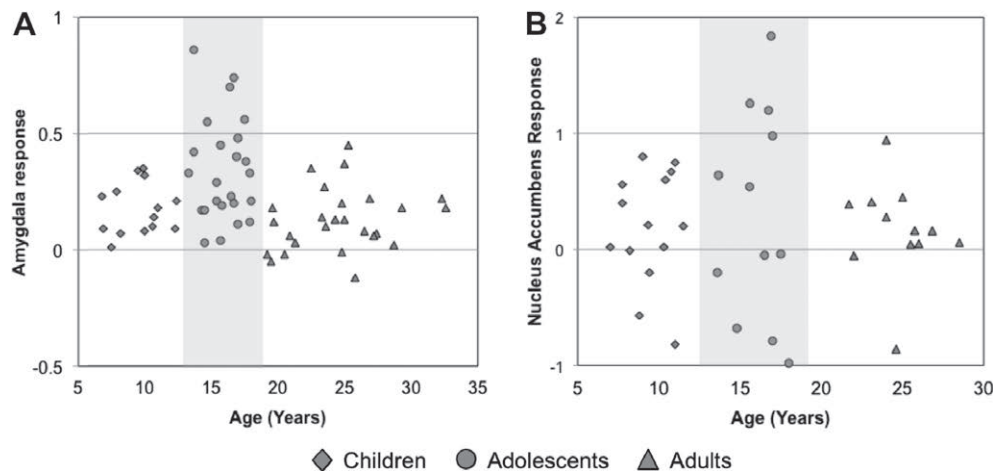


Fig. 2. (A) Amygdala response to facial expressions of emotion was significantly greater in adolescents than children or adults. Adapted from Hare et al. (2008), *Biological Psychiatry*. (B) Nucleus accumbens response to receiving a large monetary reward was significantly greater in adolescents than in children or adults. Adapted from Galvan et al. (2007), *Developmental Science*.

port to the notion that adolescents show a heightened sensitivity to the receipt of incentives, both in terms of behavior and ventral striatal responses (cf Bjork et al., 2004).

A study from our laboratory assessed changes in the neural response to appetitive cues in participants of various ages to examine neural response changes to incentives during the transition into and out of adolescence. Galvan and colleagues (2006) reported on neural responses in children, adolescents, and adults during a reward learning paradigm paying out small, medium, and large monetary incentives. In adolescents and adults, the NAcc showed linearly increasing activity as a function of reward outcome, with larger reward magnitudes eliciting greater NAcc activity. Children showed a less coordinated NAcc response, with no difference in activity across low, medium, and high reward magnitude conditions. However, in the NAcc, adolescents showed an exaggeration in this magnitude-based response, with a significant boost in response to large monetary rewards relative to children and adults (see Fig. 2B). This biological hypersensitivity to reward in adolescents has been demonstrated in several additional studies (Ernst et al., 2005; May et al., 2004) and suggests a relative functional maturity in adolescent NAcc response as compared with children, with overall patterns of response mimicking that of adults, but in an exaggerated fashion.

8. Brain mechanisms of reduced top-down control over responses to salient cues in adolescents

Another important change in brain structure occurs within tracts of white matter, bundles of myelinated axons that transport neural signals between brain regions (Cascio, Gerig, & Piven, 2007). In contrast to gray matter, white matter pathways appear to increase in size, density, and organization throughout adolescence and well into adulthood (Schmithorst, Wilke, Dardzinski, & Holland, 2002; Snook, Paulson, Roy, Phillips, & Beaulieu, 2005). Of particular interest is the structural integrity of white matter tracts between subcortical brain regions and the prefrontal cortex, as these pathways may mediate cross-communication between subcortical emotion- and incentive-driven regions and prefrontal control regions (Hare & Casey, 2005; O'Doherty, 2004; Pessoa, 2008; Phelps, 2006).

A growing body of work is accumulating to suggest that the structural integrity of subcortical–cortical white matter pathways regardless of age is related to behavior and personality characteristics pertinent to reward and emotion processing. Kim and

Whalen (in press) have recently shown that the strength of connectivity between the amygdala and the ventromedial prefrontal cortex predicts fewer symptoms of anxiety in healthy adult subjects, consistent with previous reports identifying a similar amygdala–PFC pathway (Johansen-Berg et al., 2008). Perhaps the link between structure and personality would explain individual differences in these behaviors during adolescence, where white matter maturity appears to be intermediate and variable across individuals.

Using a developmental sample, Liston and colleagues (2006) reported that several white matter tracts showed continued maturation during adolescence, including tracts between the ventral prefrontal cortex and striatum. Of the tracts examined, only the maturity of a ventral frontostriatal pathway predicted better impulse control, measured by effort in performance on a go-no-go task (Liston et al., 2006). Taken together, these studies offer intriguing evidence that subcortical–cortical white matter pathways continue to undergo structural change throughout adolescence and that the efficiency of cognitive control is, in part, dependent on the maturity of frontostriatal connections. This may be consequential to the ability to control impulses in the face of potential rewards. Future studies relating properties of white matter tracts to personality traits and cognitive abilities within developmental samples may allow greater understanding of the role of top-down and bottom-up connections in emotional- and incentive-driven behavior.

The studies discussed in the previous section suggest that adolescents may show a “hyper-reactivity” to salient environmental cues. A more comprehensive picture of adolescent emotional development takes into account the interaction between affective and control systems in the brain when required to suppress, ignore, or inhibit responses to emotional cues. Cognitive control can be defined as the ability to sustain goal-directed cognition in the face of extraneous information, and its development and neural substrates are discussed at length in another article in this volume (Luna et al., this issue). However, cognitive control is also relevant to emotional and incentive processing, because it is particularly difficult for youth to maintain cognitive control in the face of emotionally charged or incentive-laden distractors (Eigsti et al., 2006). When healthy adult participants are asked to consciously suppress their affective responses to salient environmental cues, enhanced activity is often observed in ventrolateral and medial prefrontal cortices (Ochsner & Gross, 2005; Urry et al., 2006). Counterproductive recruitment of the ventromedial prefrontal cortex may serve

as a neural predictor for psychiatric illnesses such as clinical depression (Johnstone et al., 2007), the incidence of which is elevated during adolescence. The interplay between emotional and cognitive systems is at the crux of our model, and we assert that adolescents display a functionally imbalanced pattern of neural activity that may be related to behavioral deficits in successfully inhibiting emotional responses.

More functional neuroimaging studies are needed to elucidate the interaction between emotional and controlled processing in adolescence, but initial studies have provided important insight into these interactions. A study by Monk and colleagues (2003) compared neural activity of adolescent and adult participants while they viewed fearful and neutral facial expressions of emotion. While viewing the faces, participants engaged in passive viewing rate their own emotional state. The emotional state rating was thought to necessitate shift in focus away from the facial stimuli, calling for an enhancement in controlled processes in the presence of emotion cues. Adults recruited the ventrolateral prefrontal cortex, localized to the inferior frontal gyrus to a greater extent than adolescents during trials requiring this attentional shift, when fearful faces were presented. The authors interpreted this finding as reflecting adults' ability to recruit lateral prefrontal regions to disengage from external emotional cues in order to focus on internal goals, while adolescents recruited this system less efficiently. The observation of a lateral prefrontal locus of activation is interesting and may reflect important differences between this paradigm and those presented in later sections. For example, in this experiment, activity was not correlated with any behavioral index of disengagement, implying that adolescents may be making use of different psychological strategies to complete the task at hand relative to adults. It will be important for future work to include behaviorally matched samples as well as those with modified performance across ages (presumably indexing the psychological process at hand) to further enable the interpretation of cross-developmental effects (as in Schlaggar et al. (2002)).

Hare and colleagues (2008) additionally tested for associations between subcortical and frontal regions implicated in cognitive control. Functional connectivity analyses identified a region of the ventral prefrontal cortex whose recruitment predicted the downregulation of the amygdala and less slowing of reaction times over the course of the experiment. When examining this relationship across development, adolescents under-recruited the ventral prefrontal cortex relative to adults. In other words, this study drew a linkage between under-recruitment of the ventral prefrontal cortex, exaggeration of the amygdala and slowed performance – and this pattern was characteristic of adolescents. In sum, these findings suggest that an amygdala–cortical functional network mediates the ability to exert control in the face of emotion, with adolescents showing relatively greater amygdala and differential prefrontal recruitment. This functional imbalance results in less efficiency in performing a goal-directed action in the presence of emotional cues.

Paralleling these results in the domain of incentive processing, Galvan also reported differential recruitment of the orbitofrontal cortex (OFC) in a sample including children, adolescent, and adult participants. The OFC is a subregion of the prefrontal cortex that has been shown in adults to represent reward contingencies and exert inhibitory control over risky reward-related impulses (Daw, O'Doherty, Dayan, Seymour, & Dolan, 2006; Galvan et al., 2005; see Rolls (2000) for a review). Galvan and colleagues reported that in adolescents, the OFC increased in response to the receipt of monetary reward (Galvan et al., 2006), similar to that observed in prior reports (May et al., 2004). In addition, adolescents showed spatially diffuse patterns of OFC activity that were more similar to children than adults, in contrast to the extent of activity in the NAcc, that was comparable in adolescents and adults. The spatially

diffuse activity in the OFC reported by Galvan and colleagues relative to the NAcc serves as a functional marker of brain immaturity (Durstun et al., 2006), providing additional evidence to a functional immaturity of the prefrontal cortex during the adolescent years relative to the earlier and more focal pattern of NAcc activity observed during this age.

In conclusion, subcortical systems critical to reward processing, including the ventral striatum and amygdala, show hyper-active responses to emotion and reward eliciting cues relative to both children and adults. The exaggerated neural responses in these regions lend support to the model proposed earlier, whereby amygdala and striatal signaling is disproportionately strong during the adolescent years. In contrast with the peaking of subcortical emotional and incentive-relevant brain responses, activity in the prefrontal cortex shows a very different trajectory of development. Our model theorizes that the prefrontal cortex undergoes a late-onset linear maturation with age, which is supported by structural and functional data just described. Work to date largely supports the notion that the prefrontal cortex continues to function at immature levels during the adolescent years, and exerts less regulatory control over subcortical regions relative to adults. The hyper-active upregulation of subcortical responses to salient environmental cues, paired with an immature regulatory system, may be responsible for changes in adolescent behavior, and can account for the nonlinear peak in incentive-seeking and emotional behavior often observed in adolescents.

9. Individual differences bias the responsivity of a subcortical-cortical network

The experiments just described suggest that adolescents tend to show enhanced subcortical responsivity to environmentally salient cues, as well as diminished prefrontal responses in contexts requiring cognitive control. However, simple observation of the raw data points representing the amygdala response in Fig. 2A, and nucleus accumbens response depicted in Fig. 2B, clearly shows there is substantial individual variability in these responses. In our conceptualization, adolescence in and of itself is a risk factor for the functional 'imbalance' discussed previously, but other individual difference factors may also serve as powerful mediators of subcortical-cortical responsivity (see Fig. 3). Such individual differences may take form in stable personality traits, differences in neurotransmitter profiles, biologically governed changes in hormones or other effects of puberty, and the social context, such as one's social status among peers.

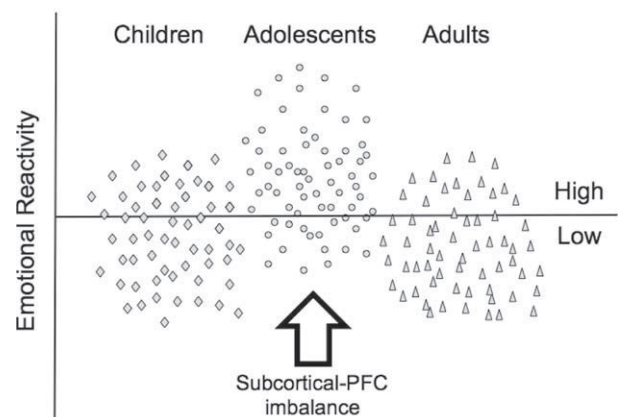


Fig. 3. Schematic representation of age and individual differences as compound risk factors for predicting highly emotional and risky behavior in adolescents.

The importance of individual differences as a predictor of 'imbalance' in subcortical–cortical networks has been demonstrated in numerous experimental contexts, including in some of the experiments described previously. Hare and colleagues (2008) showed that a substantial proportion of variability in the amygdala response to negative stimuli was accounted for by individual differences in trait anxiety irrespective of age, which is consistent with reports in adults indicating that anxiety induces a bias toward amygdala hyperresponding (Etkin et al., 2004; Somerville et al., 2004; Stein, Simmons, Feinstein, & Paulus, 2007). In terms of incentive processing, Galvan and colleagues demonstrated that across ages, a substantial proportion of variance in ventral striatal responses to the anticipation of a large reward was predicted by real-life probability of engaging in risky behavior (Galvan, Hare, Voss, Glover, & Casey, 2007). These studies offer initial evidence that individual difference variables, which are often not measured, may play an important role in biasing neural responses to affective and incentive-related cues in adolescents, and in the final sections we will examine some other additional sources of variability that may also modulate these effects. Discussion of other individual difference variables, including variability of neurotransmitter properties across development (particularly for the dopaminergic system) can be found in another article in this volume (Wahlstrom et al., this issue).

10. The role of gonadal hormones on affective and incentive processing in the adolescent brain

One potential source of influence in 'imbalanced' subcortical–cortical responding is individual differences in pubertal hormone levels. During adolescence there is a significant increase in circulating gonadal hormones, which ultimately leads to the process of sexual maturation (Spear, 2000). Gonadal hormone effects on the brain have been conceptualized into either "organizational" mechanisms whereby sex hormones cause permanent changes to neural systems which in turn influence behavior, or "activational" mechanisms whereby sex hormones only influence acute changes and the effects are reversible once the steroids are removed (Cooke, Hegstrom, Villeneuve, & Breedlove, 1998). A perspective that is becoming more common is that the acute effects of sex hormones during adolescence may sensitize neural circuits to hormone activation, which in turn allows for the development and maturation of social and sexual behaviors (Romeo, Richardson, & Sisk, 2002; Sisk & Zehr, 2005; Steinberg, 2008). In other words, adolescence may be a sensitive period for gonadal hormones to induce organizational effects, which drive social and reproductive behaviors – and potentially, emotional and incentive-seeking behaviors on a larger scale.

Sexual dimorphisms have been reported in both global changes in brain structure (Giedd, Castellanos, Rajapakse, Vaituzis, & Rapoport, 1997) as well as differing trajectories for maturation of the amygdala and striatum (Caviness, Kennedy, Richelme, Rademacher, & Filipek, 1996; Giedd et al., 1997; Schumann et al., 2004). Thus, shifts in hormonal levels may be consequential to brain development during this time of life and its associated behavioral changes. In boys (ages 8–15 years), higher basal levels of testosterone correlated with increases in volume in the amygdala (Neufang et al., 2009). This recent finding suggests that gonadal hormones may have activational effects on regions that were shown to be responsive to emotionally salient information. Because adolescence is a time when hormones levels are heightened (Norjavaara, Ankarberg, & Albertsson-Wikland, 1996), it is possible that these hormones serve as an important individual difference measure in mediating emotion and incentive-seeking behavioral and neural responses in adolescents.

Studies in adolescents also show a link between changes in hormones and social behaviors. In adolescent boys, lower levels of testosterone and testosterone levels that decreased more slowly during the day had greater levels of anxiety, depression and attention problems irrespective of pubertal development, while in adolescent girls, steeper declines in testosterone during the day correlated with greater disruptive behavior (Granger et al., 2003). In adolescent boys and girls, acute increases in gonadal hormones correlated with greater affiliations with risk-taking peers (Vermeersch, T'Sjoen, Kaufman, & Vincke, 2008a, 2008b) and higher social dominance (Schaal, Tremblay, Soussignan, & Susman, 1996) suggesting that the social environment and gonadal hormones may interact to predict individual differences in incentive and social behaviors.

While there may be a link between fluctuating hormones influencing behavior it is also important to consider the role of gonadal receptor genes, which act to mediate circulating gonadal hormones. A recent study (Perrin et al., 2008) showed variability in white matter volume in adolescent boys was mediated not only by testosterone levels but by a genetic polymorphism in the androgen receptor (AR) gene, such that boys with the short AR gene with higher testosterone levels had a greater increase in white matter volume than those with the long AR gene. This suggests the important role of genetics in understanding the activational and organization effects of hormones.

11. The influence of peers on affective and incentive processing in the adolescent brain

Relations with peers takes on a heightened importance in adolescence (Steinberg, 2005), rendering it a potential source for mediating changes in affective and incentive behavior. On one hand, adolescents as a group may show enhanced sensitivity to social cues, particularly those generated by peers, as compared to adults and children. Additionally, individual differences in sensitivity to peers may be particularly relevant in biasing adolescent behavior.

Recent studies have attempted to characterize the influence of peers on biasing behavioral and neural responses to affectively relevant cues. Grosbras et al. (2007) reported adolescents who were highly resistant to peer influence had less right dorsal premotor cortex and left dorsolateral prefrontal cortex activity while watching angry hand movements and facial expressions, versus those with lower resistance to peer influence. This suggested that individuals who are particularly sensitive to peer pressure may have an increase in motor preparation to angry movements and may engage more attention when viewing emotionally salient information. Guyer, Lau, et al. (2008) reported that female adolescents who interacted with high and low interest peers in a virtual chat room task had greater activity in the nucleus accumbens, hypothalamus, hippocampus and insula to high versus low interest peers. All of these regions, besides the insula, had age-related increases in activity suggesting a hyperresponding in reward-sensitive regions to socially desirable peers. These findings implicate the reward systems discussed earlier as potentially mediating the enhanced salience of social interactions during adolescence.

Both of these studies have attempted to elucidate the neural basis of peer influence on affective processing, yet are limited in their ability to inform neural responses during actual social interactions. In other words, during the experiments just discussed, participants do not believe they are actually interacting with peers. Work in adults has attempted to mimic real-life social interactions inside of the fMRI scanner and measure neural responses to ostensible social inclusion and exclusion (Eisenberger, Lieberman, & Williams, 2003; Somerville, Heatherton, & Kelley, 2006). Work is presently underway to develop paradigms in which adolescents are simulat-

ing or experiencing real social exchanges, and it will be of interest to assess the contribution of brain regions in reward and affective networks in mediating social behavior and monitoring the outcomes of peer interactions.

12. Caveats and limitations

The research just described, primarily conducted in just the past five years, has made remarkable strides in characterizing the nature of emotion and reward responding in the adolescent brain. However, it should be pointed out that the number of experiments on this topic is still relatively few and caution should be taken in drawing unequivocal conclusions from them. More studies with larger samples sizes are called for to fully elucidate the nature of amygdala–striatal–prefrontal interactions and their relation to adolescent behavior. In addition, testing children, adolescents, and adult subjects in a single experiment is critical for identifying nonlinear changes, because adolescents are expected to differ from both groups. This is rarely tested within a single experiment.

In terms of ventral striatal and amygdala functioning in adolescents, evidence has converged nicely in support of the idea that both systems show an exaggerated response profile in adolescents. To understand adolescent reward and emotional behavior, prefrontal control mechanisms must be taken into account, but relatively few experiments have assessed the role of the prefrontal cortex in mediating these behaviors. In addition, many experiments have discussed prefrontal responses with relative imprecision in terms of which particular area within the prefrontal cortex was active and discussing it within the context of its associated literature. The prefrontal cortex is a large area of the brain with heterogeneous subregions varying in function, architecture, inputs and outputs. Future work, both in adults and adolescents, will likely allow for greater understanding of prefrontal subdivisions and their relation to amygdala and striatal function across development.

13. Conclusions

Relative to adults and children, adolescents engage in disproportionately risky behaviors, which can lead to a wide variety of negative outcomes including substance abuse, unprotected sex, injuries, and suicide. Many of these behaviors are at least in part mediated by incentive and emotional responding, be it inappropriate appetitive behavior leading to risky approach of potential rewards, or the outcome of experiencing extreme negative affect such as self-harm and suicide. Emotional and incentive-related behaviors are intimately linked to these risks, and understanding the role of developing brain systems in mediating these behaviors is of inherent importance to adolescent health.

Human structural and functional imaging studies have begun to shed light on the complex changes occurring in the brain at this time of life, and their relationship to adolescent behavior. At this point, it appears that the differential trajectories of the amygdala and nucleus accumbens, relative to late-maturing control regions in the prefrontal cortex, may lead to adolescent behavioral changes characterized by enhanced sensitivity to environmental cues without appropriate behavioral inhibition. A host of individual differences also appear to be critical for predicting heightened risk for this behavioral profile, which are just beginning to be explored empirically. Relatively mature emotional and reward systems left unchecked by prefrontal control systems may be the key neural ‘imbalance’ that leads to the nonlinear, unique behavioral profile of adolescents. It is hoped that continued work in this field will improve our understanding of this fascinating and complex time of life.

Acknowledgments

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References

- Abe, K., & Suzuki, T. (1986). Prevalence of some symptoms in adolescence and maturity: Social phobias, anxiety symptoms, episodic illusions and ideas of reference. *Psychopathology*, 19, 200–205.
- Adolphs, R. (2002). Recognizing emotion from facial expressions: Psychological and neurological mechanisms. *Behavioral and Cognitive Neuroscience Reviews*, 1(1), 21–62.
- Adriani, W., Chiarotti, F., & Laviola, G. (1998). Elevated novelty seeking and peculiar d-amphetamine sensitization in periadolescent mice compared with adult mice. *Behavioral Neuroscience*, 112(5), 1152–1166.
- Aggleton, J. P. (2000). *The amygdala: A functional analysis*. New York: Oxford University Press.
- Arnett, J. (1999). Adolescent storm and stress, reconsidered. *American Psychologist*, 54, 317–326.
- Baird, A. A., Gruber, S. A., Fein, D. A., Maas, L. C., Steingard, R. J., Renshaw, P. F., et al. (1999). Functional magnetic resonance imaging of facial affect recognition in children and adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry*, 38(2), 195–199.
- Bjork, J. M., Knutson, B., Fong, G. W., Caggiano, D. M., Bennett, S. M., & Hommer, D. W. (2004). Incentive-elicited brain activation in adolescents: Similarities and differences from young adults. *Journal of Neuroscience*, 24(8), 1793–1802.
- Blakemore, S.-J. (2008). The social brain in adolescence. *Nature Reviews Neuroscience*, 9, 267–277.
- Bogin, B. (1994). Adolescence in evolutionary perspective. *Acta Paediatrica Supplement*, 406, 29–35.
- Buchanan, C. M., Eccles, J. S., & Becker, J. B. (1992). Are adolescents the victims of raging hormones: Evidence for activation effects of hormones on moods and behavior at adolescence. *Psychological Bulletin*, 111, 62–107.
- Cardinal, R. N., Parkinson, J. A., Hall, J., & Everitt, B. J. (2002). Emotion and motivation: The role of the amygdala, ventral striatum, and prefrontal cortex. *Neuroscience and Biobehavioral Reviews*, 26(3), 321–352.
- Cascio, C. J., Gerig, G., & Piven, J. (2007). Diffusion tensor imaging: Application to the study of the developing brain. *Journal of the American Academy of Child and Adolescent Psychiatry*, 46(2), 213–223.
- Casey, B. J., Galvan, A., & Hare, T. A. (2005). Changes in cerebral functional organization during cognitive development. *Current Opinion in Neurobiology*, 15(2), 239–244.
- Casey, B. J., Getz, S., & Galvan, A. (2008a). The adolescent brain. *Developmental Review*, 28(1), 62–77.
- Casey, B. J., Jones, R. M., & Hare, T. (2008b). The adolescent brain. *Annals of the New York Academy of Sciences*, 1124, 111–126.
- Casey, B. J., Tottenham, N., Liston, C., & Durston, S. (2005). Imaging the developing brain: What have we learned about cognitive development? *Trends in Cognitive Science*, 9(3), 104–110.
- Cauffman, E., Shulman, E. P., Steinberg, L., Claus, E., Banich, M. T., Graham, S. J., et al. (in press). Age differences in affective decision making as indexed by performance on the Iowa Gambling Task. *Developmental Psychology*.
- Caviness, V. S., Kennedy, D. N., Richelme, C., Rademacher, J., & Filipek, P. A. (1996). The human brain age 7–11 years: A volumetric analysis based on magnetic resonance images. *Cerebral Cortex*, 6, 726–736.
- Compas, B. E., Hinden, B. R., & Gerhardt, C. A. (1995). Adolescent development: Pathways and processes of risk and resilience. *Annual Review of Psychology*, 46, 265–293.
- Compas, B. E., Orosan, P. G., & Grant, K. E. (1993). Adolescent stress and coping: Implications for psychopathology during adolescence. *Journal of Adolescence*, 16, 331–349.
- Cooke, B., Hegstrom, C. D., Villeneuve, L. S., & Breedlove, S. M. (1998). Sexual differentiation of the vertebrate brain: Principles and mechanisms. *Frontiers in Neuroendocrinology*, 19(4), 323–362.
- Davis, M., & Whalen, P. J. (2001). The amygdala: Vigilance and emotion. *Molecular Psychiatry*, 6(1), 13–34.
- Daw, N. D., O'Doherty, J. P., Dayan, P., Seymour, B., & Dolan, R. J. (2006). Cortical substrates for exploratory decisions in humans. *Nature*, 441, 876–879.
- Delgado, M. R. (2007). Reward-related responses in the human striatum. *Annals of the New York Academy of Sciences*, 1104, 70–88.
- Delgado, M. R., Nystrom, L. E., Fissell, C., Noll, D. C., & Fiez, J. A. (2000). Tracking the hemodynamic responses to reward and punishment in the striatum. *Journal of Neurophysiology*, 84(6), 3072–3077.
- Durston, S., Davidson, M. C., Tottenham, N., Galvan, A., Spicer, J., Fossella, J. A., et al. (2006). A shift from diffuse to focal cortical activity with development. *Developmental Science*, 9(1), 1–8.
- Eaton, L. K., Kann, L., Kinchen, S., Shanklin, S., Ross, J., Hawkins, J., et al. (2008). Youth risk behavior surveillance – United States, 2007, surveillance summaries. *Morbidity and Mortality Weekly Report*, 57(SS04), 1–131.
- Eccles, J. S., Wigfield, A., Flanagan, C. A., Miller, C., Reuman, D. A., & Yee, D. (1989). Self concepts, domain values, and self-esteem: Relations and changes at early adolescence. *Journal of Personality*, 57, 283–310.

- Eigsti, I. M., Zayas, V., Mischel, W., Shoda, Y., Ayduk, O., Dadlani, M. B., et al. (2006). Predicting cognitive control from preschool to late adolescence and young adulthood. *Psychological Science*, 17(6), 478–484.
- Eisenberger, N. I., Lieberman, M. D., & Williams, K. D. (2003). Does rejection hurt? An fMRI study of social exclusion. *Science*, 302(5643), 290–292.
- Ernst, M., Nelson, E. E., Jazbec, S., McClure, E. B., Monk, C. S., Leibenluft, E., et al. (2005). Amygdala and nucleus accumbens in responses to receipt and omission of gains in adults and adolescents. *Neuroimage*, 25(4), 1279–1291.
- Ernst, M., Pine, D. S., & Hardin, M. (2006). Triadic model of the neurobiology of motivated behavior in adolescence. *Psychological Medicine*, 36(3), 299–312.
- Etkin, A., Klemmehagen, K. C., Dudman, J. T., Rogan, M. T., Hen, R., Kandel, E. R., et al. (2004). Individual differences in trait anxiety predict the response of the basolateral amygdala to unconsciously processed fearful faces. *Neuron*, 44(6), 1043–1055.
- Fareri, D. S., Martin, L. N., & Delgado, M. R. (2008). Reward-related processing in the human brain: Developmental considerations. *Development and Psychopathology*, 20, 1191–1211.
- Figner, B., Mackinlay, R. J., Wilkening, F., & Weber, E. U. (2009a). Affective and deliberative processes in risky choice: Age differences in risk taking in the Columbia Card Task. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, 35(3), 709–730.
- Figner, B., Mackinlay, R. J., Wilkening, F., & Weber, E. U. (2009b). Risky choice in children, adolescents, and adults: Affective versus deliberative processes and the role of executive functions. In *Proceedings of the society for research in child development*, Denver, CO, USA.
- Galvan, A., Hare, T. A., Davidson, M., Spicer, J., Glover, G., & Casey, B. J. (2005). The role of ventral frontostriatal circuitry in reward-based learning in humans. *Journal of Neuroscience*, 25(38), 8650–8656.
- Galvan, A., Hare, T. A., Parra, C. E., Penn, J., Voss, H., Glover, G., et al. (2006). Earlier development of the accumbens relative to orbitofrontal cortex might underlie risk-taking behavior in adolescents. *Journal of Neuroscience*, 26(25), 6885–6892.
- Galvan, A., Hare, T., Voss, H., Glover, G., & Casey, B. J. (2007). Risk-taking and the adolescent brain: Who is at risk? *Developmental Science*, 10(2), F8–F14.
- Gardner, M., & Steinberg, L. (2005). Peer influence on risk taking, risk preference, and risky decision making in adolescence and adulthood: An experimental study. *Developmental Psychology*, 41, 625–635.
- Giedd, J. N., Castellanos, F. X., Rajapakse, J. C., Vaituzis, A. C., & Rapoport, J. L. (1997). Sexual dimorphism of the developing human brain. *Progress in Neuropsychopharmacology and Biological Psychiatry*, 21(8), 1185–1201.
- Giedd, J. N., Vaituzis, A. C., Hamburger, S. D., Lange, N., Rajapakse, J. C., Kayser, D., et al. (1996). Quantitative MRI of the temporal lobe, amygdala, and hippocampus in normal human development: Ages 4–18 years. *The Journal of Comparative Neurology*, 366, 223–230.
- Giedd, J. N., Blumenthal, J., Jeffries, N. O., Castellanos, F. X., Liu, H., Zijdenbos, A., et al. (1999). Brain development during childhood and adolescence: A longitudinal MRI study. *Nature Neuroscience*, 2, 861–863.
- Granger, D. A., Shirtcliff, E. A., Zahn-Waxler, C., Usher, B., Klimes-Dougan, B., & Hastings, P. (2003). Salivary testosterone diurnal variation and psychopathology in adolescent males and females: Individual differences and developmental effects. *Developmental Psychopathology*, 15(2), 431–449.
- Grosbras, M.-H., Jansen, M., Leonard, G., McIntosh, A., Osswald, K., Poulsen, C., et al. (2007). Neural mechanisms of resistance to peer influence in early adolescence. *Journal of Neuroscience*, 27(30), 8040–8045.
- Guyer, A. E., Lau, J. Y., McClure-Tone, E. B., Parrish, J., Shiffrin, N. D., Reynolds, R. C., et al. (2008b). Amygdala and ventrolateral prefrontal cortex function during anticipated peer evaluation in pediatric social anxiety. *Archives of General Psychiatry*, 65(11), 1303–1312.
- Guyer, A. E., Monk, C. S., McClure-Tone, E. B., Nelson, E. E., Roberson-Nay, R., Adler, A., et al. (2008a). A developmental examination of amygdala response to facial expressions. *Journal of Cognitive Neuroscience*, 20(9), 1565–1582.
- Hall, G. S. (1904). *Adolescence: In psychology and its relation to physiology, anthropology, sociology, sex, crime, religion, and education* (Vols. I and II). Englewood Cliffs, NJ: Prentice-Hall.
- Hare, T. A., & Casey, B. J. (2005). The neurobiology and development of cognitive and affective control. *Cognition, Brain and Behavior*, 9(3), 273–286.
- Hare, T. A., Tottenham, N., Galvan, A., Voss, H. U., Glover, G. H., & Casey, B. J. (2008). Biological substrates of emotional reactivity and regulation in adolescence during an emotional go-nogo task. *Biological Psychiatry*, 63(10), 927–934.
- Huttenlocher, P. R. (1997). Regional differences in synaptogenesis in human cerebral cortex. *Journal of Comparative Neurology*, 387, 167–178.
- Johansen-Berg, H., Gutman, D. A., Behrens, T. E. J., Matthews, P. M., Rushworth, M. F. S., Katz, E., et al. (2008). Anatomical connectivity of the subgenual cingulate region targeted with deep brain stimulation for treatment-resistant depression. *Cerebral Cortex*, 18, 1374–1383.
- Kessler, R. C., Berglund, P., Delmer, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, 62, 593–602.
- Kim, M. J., & Whalen, P. J. The structural integrity of an amygdala-prefrontal pathway predicts trait anxiety. *Journal of Neuroscience*.
- Knutson, B., Adams, C. M., Fong, G. W., & Hommer, D. (2001). Anticipation of increasing monetary reward selectively recruits nucleus accumbens. *Journal of Neuroscience*, 21(16), RC159.
- Kober, H., Barrett, L. F., Joseph, J., Bliss-Moreau, E., Lindquist, K., & Wager, T. D. (2008). Functional grouping and cortical-subcortical interactions in emotion: A meta-analysis of neuroimaging studies. *Neuroimage*, 42(2), 998–1031.
- Larson, R. W., Moneta, G., Richards, M. H., & Wilson, S. (2002). Continuity, stability, and change in daily emotional experience across adolescence. *Child Development*, 73(4), 1151–1165.
- Laviola, G., Macri, S., Morley-Fletcher, S., & Adriani, W. (2003). Risk-taking behavior in adolescent mice: Psychobiological determinants and early epigenetic influence. *Neuroscience Biobehavioral Review*, 27(1–2), 19–31.
- LeDoux, J. E. (2000). Emotion circuits in the brain. *Annual Review of Neuroscience*, 23, 155–184.
- Liston, C., Watts, R., Tottenham, N., Davidson, M. C., Niogi, S., Ulug, A. M., et al. (2006). Frontostriatal microstructure modulates efficient recruitment of cognitive control. *Cerebral Cortex*, 16(4), 553–560.
- May, J. C., Delgado, M. R., Dahl, R. E., Stenger, V. A., Ryan, N. D., Fiez, J. A., et al. (2004). Event-related functional magnetic resonance imaging of reward-related brain circuitry in children and adolescents. *Biological Psychiatry*, 55(4), 359–366.
- Miller, E. K., & Cohen, J. D. (2001). An integrative theory of prefrontal cortex function. *Annual Review of Neuroscience*, 24, 167–202.
- Monk, C. S., McClure, E. B., Nelson, E. E., Zarah, E., Bilder, R. M., Leibenluft, E., et al. (2006). Adolescent immaturity in attention-related brain engagement to emotional facial expressions. *Neuroimage*, 20, 420–428.
- Mościcki, E. (2001). Epidemiology of attempted and completed suicide: Toward a framework for prevention. *Clinical Neuroscience Research*, 1, 310–323.
- National Research Council (2007). *Preventing teen motor crashes: Contributions from the behavioral and social sciences*. Washington, DC: National Academies Press.
- Nelson, E. E., Leibenluft, E., McClure, E. B., & Pine, D. S. (2005). The social re-orientation of adolescence: A neuroscience perspective on the process and its relation to psychopathology. *Psychological Medicine*, 35, 163–174.
- Neufang, S., Specht, K., Hausmann, M., Gunturkun, O., Herpertz-Dahlmann, B., Fink, G. R., et al. (2009). Sex differences and the impact of steroid hormones on the developing human brain. *Cerebral Cortex*, 19(2), 464–473.
- Norjavaara, E., Ankarberg, C., & Albertsson-Wikland, K. (1996). Diurnal rhythm of 17 beta-estradiol secretion throughout pubertal development in healthy girls: Evaluation by a sensitive radioimmunoassay. *Journal of Clinical Endocrinology and Metabolism*, 81(11), 4095–4102.
- O'Doherty, J. P. (2004). Reward representations and reward-related learning in the human brain: Insights from neuroimaging. *Current Opinion in Neurobiology*, 14(6), 769–776.
- O'Doherty, J. P., Deichmann, R., Critchley, H. D., & Dolan, R. J. (2002). Neural responses during anticipation of a primary taste reward. *Neuron*, 33(5), 815–826.
- Ochsner, K. N., & Gross, J. J. (2005). The cognitive control of emotion. *Trends in Cognitive Science*, 9(5), 242–249.
- Paus, T., Keshavan, M., & Giedd, J. N. (2008). Why do many psychiatric disorders emerge during adolescence? *Nature Reviews Neuroscience*, 9, 947–957.
- Perrin, J. S., Herve, P. Y., Leonard, G., Perron, M., Pike, G. B., Pitiot, A., et al. (2008). Growth of white matter in the adolescent brain: Role of testosterone and androgen receptor. *Journal of Neuroscience*, 28(38), 9519–9524.
- Pessoa, L. (2008). On the relationship between emotion and cognition. *Nature Reviews Neuroscience*, 9(2).
- Petersen, A. C., Compas, B. E., Brooks-Gunn, J., Stemmler, M., Ey, S., & Grant, K. E. (1993). Depression in adolescence. *American Psychologist*, 48, 155–168.
- Phelps, E. A. (2006). Emotion and cognition: Insights from studies of the human amygdala. *Annual Review of Psychology*, 57, 27–53.
- Pine, D. S., Cohen, P., & Brook, J. S. (2001). Emotional reactivity and risk for psychopathology among adolescents. *CNS Spectrum*, 6(1), 27–35.
- Rakic, P., Bourgeois, J. P., Eckenhoff, M. F., Zecevic, N., & Goldman-Rakic, P. S. (1986). Concurrent overproduction of synapses in diverse regions of the primate cerebral cortex. *Science*, 232, 232–235.
- Reyna, V. F., & Farley, F. (2006). Risk and rationality in adolescent decision making: Implications for theory, practice, and public policy. *Psychological Science in the Public Interest*, 7(1), 1–44.
- Rolls, E. (2000). The orbitofrontal cortex and reward. *Cerebral Cortex*, 10, 284–294.
- Romeo, R. D., Richardson, H. N., & Sisk, C. L. (2002). Puberty and the maturation of the male brain and sexual behavior: Recasting a behavioral potential. *Neuroscience and Biobehavioral Reviews*, 26(3), 381–391.
- Rutter, M., Graham, P., Chadwick, O. F. D., & Yule, W. (1976). Adolescent turmoil: Fact or fiction? *Journal of Child Psychology and Psychiatry*, 17, 35–56.
- Schaal, B., Tremblay, R. E., Soussignan, R., & Susman, E. J. (1996). Male testosterone linked to high social dominance but low physical aggression in early adolescence. *Journal of the American Academy of Child and Adolescent Psychiatry*, 35(10), 1322–1330.
- Schlaggar, B. L., Brown, T. T., Lugar, H. M., Visscher, K. M., Miezin, F. M., & Petersen, S. E. (2002). Functional neuroanatomical differences between adults and school-age children in the processing of single words. *Science*, 296(5572), 1476–1479.
- Schmithorst, V. J., Wilke, M., Dardzinski, B. J., & Holland, S. K. (2002). Correlation of white matter diffusivity and anisotropy with age during childhood and adolescence: A cross-sectional diffusion-tensor MR imaging study. *Radiology*, 222, 212–218.
- Schultz, W. (2006). Behavioral theories and the neurophysiology of reward. *Annual Reviews of Psychology*, 57, 87–115.
- Schultz, W., Dayan, P., & Montague, P. R. (1997). A neural substrate of prediction and reward. *Science*, 275(5306), 1593–1599.
- Schumann, C. M., JHammstra, J., Goodlin-Jones, B. L., Lotspeich, L. J., Kwon, H., Buonocore, M. H., et al. (2004). The amygdala is enlarged in children but not adolescents with autism; the hippocampus is enlarged at all ages. *Journal of Neuroscience*, 24(28), 6392–6401.

- Silveri, M. M., Tzilos, G. K., Pimentel, P. J., & Yurgelun-Todd, D. A. (2004). Trajectories of adolescent emotional and cognitive development: Effects of sex and risk for drug use. *Annals of the New York Academy of Sciences*, 1021, 363–370.
- Simmons, R. G., & Blyth, D. A. (1987). *Moving into adolescence: The impact of pubertal change and school context*. Hawthorne, NY: Aldine de Gruyter.
- Simmons, R. G., Rosenberg, F., & Rosenberg, M. (1973). Disturbance in the self-image at adolescence. *American Sociological Review*, 38, 553–568.
- Sisk, C. L., & Zehr, J. L. (2005). Pubertal hormones organize the adolescent brain and behavior. *Frontiers in Neuroendocrinology*, 26(3–4), 163–174.
- Snook, L., Paulson, L. A., Roy, D., Phillips, L., & Beaulieu, C. (2005). Diffusion tensor imaging of neurodevelopment in children and young adults. *Neuroimage*, 26, 1164–1173.
- Somerville, L. H., Fani, N., & McClure-Tone, E. B. (in press). Behavioral and neural representations of emotional facial expressions across the lifespan. *Developmental Neuropsychology*.
- Somerville, L. H., Heatherton, T. F., & Kelley, W. M. (2006). Anterior cingulate cortex responds differentially to expectancy violation and social rejection. *Nature Neuroscience*, 9(8), 1007–1008.
- Somerville, L. H., Kim, H., Johnstone, T., Alexander, A. L., & Whalen, P. J. (2004). Human amygdala responses during presentation of happy and neutral faces: Correlations with state anxiety. *Biological Psychiatry*, 55(9), 897–903.
- Sowell, E. R., Trauner, D. A., Gamst, A., & Jernigan, T. L. (2002). Development of cortical and subcortical brain areas in childhood and adolescence: A structural MRI study. *Developmental Medicine and Child Neurology*, 44(1), 4–16.
- Spear, L. P. (2000). The adolescent brain and age-related behavioral manifestations. *Neuroscience and Biobehavioral Reviews*, 24(4), 417–463.
- Stein, M. B., Simmons, A. N., Feinstein, J. S., & Paulus, M. P. (2007). Increased amygdala and insula activation during emotion processing in anxiety-prone subjects. *American Journal of Psychiatry*, 164(2), 318–327.
- Steinberg, L. (2004). Risk taking in adolescence: What changes, and why? *Annals of the New York Academy of Sciences*, 1021, 51–58.
- Steinberg, L. (2005). Cognitive and affective development in adolescence. *Trends in Cognitive Sciences*, 9(2), 69–74.
- Steinberg, L. (2008). A social neuroscience perspective on adolescent risk-taking. *Developmental Review*, 28, 78–106.
- Substance Abuse and Mental Health Services Administration (2007). Results from the 2006 national survey on drug use and health: National findings. Office of Applied Studies NSDUH Series H32, Publication No. SMA 07-4293. Rockville, MD.
- Thomas, K. M., Drevets, W. C., Whalen, P. J., Eccard, C. H., Dahl, R. E., Ryan, N. D., et al. (2001). Amygdala response to facial expressions in children and adults. *Biological Psychiatry*, 49, 309–316.
- Thornburg, H. D., & Jones, R. M. (1982). Social characteristics of early adolescents: Age vs. grade. *Journal of Early Adolescence*, 2, 229–239.
- Urry, H. L., van Reekum, C. M., Johnstone, T., Kalin, N. H., Thuro, M. E., Schaefer, H. S., et al. (2006). Amygdala and ventromedial prefrontal cortex are inversely coupled during regulation of negative affect and predict diurnal pattern of cortisol secretion among older adults. *Journal of Neuroscience*, 26(16), 4415–4425.
- Vermeersch, H., T'Sjoen, G., Kaufman, J. M., & Vincke, J. (2008a). The role of testosterone in aggressive and non-aggressive risk-taking in adolescent boys. *Hormones and Behavior*, 53(3), 463–471.
- Vermeersch, H., T'Sjoen, G., Kaufman, J. M., & Vincke, J. (2008b). Estradiol, testosterone, differential association and aggressive and non-aggressive risk-taking in adolescent girls. *Psychoneuroendocrinology*, 33(7), 897–908.
- Williams, L. M., Brown, K. J., Palmer, D., Liddell, B. J., Kemp, A. H., Olivieri, G., et al. (2006). The mellow years? Neural basis of improving emotional stability with age. *Journal of Neuroscience*, 26(24), 6422–6430.
- Zuckerman, M., Eysenck, S., & Eysenck, H. J. (1978). Sensation seeking in England and America: Cross-cultural, age, and sex comparisons. *Journal of Consulting and Clinical Psychology*, 46, 139–149.

EXHIBIT 3

EXCERPT

The Case for Gun Policy Reforms in America



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*On February 5, 2014, this White Paper was updated to reflect a correction concerning the number of states that allow 18-20 year-olds to legally purchase handguns (page 5).



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The Burden of Gun Violence in the United States

More than 31,000 people a year in the United States die from gunshot wounds.¹ Because victims are disproportionately young, gun violence is one of the leading causes of premature mortality in the U.S. In addition to these deaths, in 2010, there were an estimated 337,960 non-fatal violent crimes committed with guns,² and 73,505 persons treated in hospital emergency departments for non-fatal gunshot wounds.^{3,4}

Gun violence in the United States is unusually high for a nation of such wealth. Although there is little difference in the overall crime rates between the United States and other high-income countries, the homicide rate in the U.S. is seven times higher than the combined homicide rate of 22 other high-income countries.⁵ This is because the *firearm* homicide rate in the U.S. is twenty times greater than in these other high-income countries. The higher prevalence of gun ownership and much less restrictive gun laws are important reasons why violent crime in the U.S. is so much more lethal than in countries of similar income levels.

There are enormous economic costs associated with gun violence in the U.S. Firearm-related deaths and injuries resulted in medical and lost productivity expenses of about \$32 billion in 2005.⁶ But the overall cost of gun violence goes well beyond these figures. When lost quality of life, psychological and emotional trauma, decline in property values, and other legal and societal consequences are included, the cost of gun violence in the U.S. was estimated to be about \$100 billion annually in 1998.⁷ A new study has examined the direct and indirect costs of violent crime in eight geographically-diverse U.S. cities, and estimated the average annual cost of violent crime was more than \$1,300 for every adult and child. Because much of these costs are due to lowering residential property values, violent crime greatly reduces tax revenues that local governments need to address a broad array of citizens' needs. The direct annual cost of violent crime to all levels of government was estimated to be \$325 per resident.⁸

Gun Control Policies in the United States

Debates about gun control often drift towards general arguments about whether guns make us safer or less safe, and gun control is equated with restricting gun ownership. However, with recent Supreme Court decisions overturning laws which ban firearm possession in the District of Columbia and Chicago, current gun control policies in the U.S. do not disarm law-abiding adults over the age of 21. Rather, gun control laws today focus on one or more of four general objectives. These laws aim to:

1. Define conditions that prohibit a person from possessing firearms;
2. Implement regulations to prevent prohibited persons from possessing firearms;
3. Restrict carrying of concealed firearms outside the home; and
4. Regulate the design of firearms to enhance public and personal safety.

Below, we draw upon research evidence to suggest how improvements in each of these types of gun policies could enhance public safety in the United States.

Rationale for Current Conditions that Prohibit Firearm Possession

Federal law prohibits certain categories of individuals from possessing firearms, including: felons; fugitives; persons convicted of a misdemeanor crime for domestic violence; those who are subject to certain restraining orders for domestic violence; unlawful users of or those addicted to controlled substances; those who have been found by a judge to be mentally incompetent, a danger to themselves or others as a result of mental illness, or been involuntarily committed to a mental institution; those who have been dishonorably discharged from the military; illegal aliens; and persons who have renounced their U.S. citizenship. In addition, federal law sets 21 years as the minimum age at which a person can lawfully purchase a handgun from a federally licensed firearms dealer, but sets 18 as the minimum legal age for handgun possession and for transfers of handguns from anyone who is not a licensed gun dealer.⁹

Most of these categories of persons prohibited from possessing firearms can be justified based on data indicating increased risk for violence. Individuals with prior felony convictions are far more likely to commit future crimes of violence than non-felons. A history of perpetrating intimate partner violence (IPV) is associated with increased risk of subsequent murder of an intimate partner, and a perpetrator's ownership of a firearm increases the risk of domestic homicide five-fold for victims.¹⁰ Several studies indicate that a significant proportion of domestic violence abusers also commit serious offenses against strangers and non-family members.^{11,12,13,14}

Firearm prohibitions for drug abusers are also justified. Substance abuse is associated with increased risk of domestic violence,^{15,16,17,18} and incarceration for violent crime,¹⁹ as well as suicide.^{20,21} Homicide offenders are nearly five times more likely to abuse drugs than non-offender controls.²² Although the majority of persons with mental illnesses are not violent,^{23,24} and only a small portion of violence is attributable to mental illness alone,²⁵ persons with serious mental illnesses, such as schizophrenia, bipolar disorder, and major depression, are more likely to commit violence against others and themselves than are individuals who do not have these disorders.^{26,27, 28}

Minimum age restrictions for firearm possession are prudent because the risk of perpetrating or being victimized by serious violent crimes increases rapidly during adolescence and in the early 20s.^{29, 30} (See Figure below.) Brain structures related to risk-taking and impulse-control are developing throughout adolescence, and this may contribute to heightened risk of violent behavior among this age group.^{31,32}

Why Firearms Prohibitions for High-Risk Persons Should be Broadened

Criminal Prohibitions

In addition to the exclusion criteria for firearm possession under federal law, many states have additional disqualifications for legal firearm possession. The differences in exclusion criteria across states are significant. For example, New Jersey prohibits firearm possession by anyone who has been convicted of a crime for which the penalty can be 6 months or more of

imprisonment, and sets the minimum legal age for handgun possession at 21 years. In contrast, 13 states have standards for legal firearm possession that either mirror or are weaker than federal standards.

Most people believe that criminals should not be able to possess firearms lawfully. Yet, our current laws permit many people who have been convicted of crimes—most misdemeanor crimes adjudicated in adult court and felony crimes handled in juvenile court—to possess firearms. Data from two studies of individuals who have committed the most serious crimes indicate that prior to committing these crimes, the perpetrators were not prohibited from possessing a firearm under federal law. A recent study, based on surveys of inmates in state prisons, examined the criminal history and ages of persons imprisoned for committing crimes with a gun in the 13 states with standards for legal gun ownership that do not go beyond those set in federal law. At the time when they committed the gun crime leading to their incarceration, only 27 percent of these gun offenders were prohibited from possessing firearms because they had previously been convicted of a felony. Of these offenders, 60 percent could legally possess guns prior to committing the gun crime that led to their incarceration, including four percent who had prior misdemeanor convictions involving violence and/or firearms, six percent convicted of other misdemeanors, five percent convicted of a felony in a juvenile court, and 13 percent with prior arrests but no convictions.³³

Some may assume that persons convicted of misdemeanor crimes do not pose a significant threat for committing serious violent crimes. But many suspects charged with felony crimes are convicted of lesser charges as a result of a plea agreement. Research has shown that misdemeanants who were legally able to purchase handguns committed crimes involving violence following those purchases at a rate two to ten times higher than that of handgun purchasers with no prior convictions.³⁴ Handgun purchasers with a history of arrest but no convictions have an equally high or higher risk of committing violent crimes following handgun purchases as do misdemeanants who legally purchased a handgun.³⁵

We believe the evidence above justifies an extension of firearm prohibitions for persons with a history of criminal behavior to include persons convicted of all misdemeanor crimes of violence, as well as individuals who have committed felony crimes as a juvenile. Such prohibitions do not necessarily need to be life-long. Many states have laws prohibiting firearm possession for individuals convicted of serious crimes as juveniles. These restrictions are time-limited, based on either the age of the individual or the number of years since the prohibiting conviction.

Substance Abusers

Federal law prohibits firearm possession by anyone who is addicted to illegal drugs, but regulations written to implement the law provide a relatively narrow definition of who would be prohibited. A person would be determined to be prohibited if he has “a conviction for use or possession of a controlled substance within the past year; multiple arrests for such offenses within the past 5 years, if the most recent arrest occurred within the past year; or ... [is] found through a drug test to use a controlled substance unlawfully, provided the test was administered within the past year.”³⁶ The number of drug abusers prohibited from possessing firearms might

be increased significantly by revamping these regulations to, for example, expand the period following a drug conviction for which a person is prohibited from possessing firearms.

Expanding firearm prohibitions to include persons who are alcoholics or problem drinkers could potentially reduce alcohol-related violence. Alcohol abuse is strongly associated with the perpetration of violence. Yet federal firearm laws do not prohibit alcoholics from possessing firearms, and only 16 states have statutes prohibiting alcohol abusers from possessing firearms. Furthermore, some states with gun prohibitions for alcohol abusers lack regulations to allow authorities to enforce the prohibition.³⁷

Youth Under Age 21

Restrictions on youths' ability to purchase and possess firearms should be broadened. Although federal law and most state law allows youth 18 to 20 years of age to legally possess a handgun, youth of these ages have some of the highest rates of homicide offending. Age-specific homicide offending rates rise sharply in the late teens and peak at age 20.³⁸ (See figure below.) In an examination of the background and legal status of gun offenders incarcerated in the 13 states with the weakest standards for legal firearm ownership, the largest segment of offenders who would have been prohibited in other states with stricter standards were those who were between 18 and 20 years of age.³³ Heightened risk-taking, and concerns for protecting youth and the public from alcohol abuse resulted in laws in all 50 states, establishing 21 as the minimum legal age for alcoholic beverage consumption. These laws led to significant reductions in deaths from motor vehicle crashes involving drivers ages 18-20.³⁹ Yet, *thirty-eight states allow 18- to 20-year-olds to legally possess as many handguns as they desire.

Homicide offending per 100,000 population in 2009 for specific ages of offenders.*



* Data from the Supplemental Homicide Reports, Uniform Crime Reporting System, Federal Bureau of Investigation, U.S. Department of Justice.

*On February 5, 2014, this White Paper was updated to reflect the following correction: Thirty-eight states allow 18-20 year olds to legally possess as many handguns as they desire. An earlier version incorrectly reported forty-five states allowed 18-20 year olds to legally possess as many handguns as they desire.

Conclusion

The burden of gun violence on American society is substantial, whether measured in years of productive life lost, disability, fear, or economic costs. The toll is unprecedented among high-income nations. Weaknesses in current gun laws contribute to this burden by establishing low standards for legal gun ownership and significant loopholes in policies designed to keep guns from prohibited persons. When states expand firearm prohibitions to high-risk groups, and adopt comprehensive measures to prevent diversion of guns to prohibited persons, fewer guns are diverted to criminals, and there is less violence.

Some mistakenly believe that the Second Amendment to the U.S. Constitution would prohibit the kinds of legal reforms we believe are warranted. In 2008, in *District of Columbia v. Heller*,⁹¹ the U.S. Supreme Court ruled that the Second Amendment protected an individual right to own guns, striking down Washington, D.C.'s law banning handgun possession in the home. However, the *Heller* decision also mentioned numerous types of presumptively valid gun laws, including "laws imposing conditions and qualifications on the commercial sale of arms." Since *Heller*, lower courts have overwhelmingly upheld the constitutionality of a wide range of gun laws other than handgun bans.⁹²

Contrary to recent media reports, a large majority of the public, including gun owners, favors remedying many current weaknesses in our gun laws. There are real political hurdles to enacting new gun control laws, and the power of the gun lobby is substantial. But politicians who want to correct flaws in our current laws, which enable dangerous people to get guns, could do so knowing that there is broad support for those policies, the reforms are constitutional, and the policies would enhance public safety.

References

¹ Centers for Disease Control and Prevention. Web-based Injury Statistics Query and Reporting System (WISQARS) [Online]. National Center for Injury Control and Prevention, Centers for Disease Control and Prevention (producer). Available from: URL: <http://www.cdc.gov/injury/wisqars/index.html>. [2012, Mar. 15].

² Truman JL. Criminal Victimization, 2010. National Crime Victimization Survey. NCJ 235508, Washington, DC: United States Department of Justice, Bureau of Justice Statistics, Sept. 2010.

³ Centers for Disease Control and Prevention. Web-based Injury Statistics Query and Reporting System (WISQARS) [Online]. National Center for Injury Control and Prevention, Centers for Disease Control and Prevention (producer). Available from: URL: <http://www.cdc.gov/injury/wisqars/index.html>. [2012, Mar. 15].

⁴ Vyrostek SB, Annest JL, Ryan GW. Surveillance for Fatal and Non-Fatal Injuries – United States, 2001. *MMWR*. 2004; 53(SS07):1-57.

⁵ Richardson EG, Hemenway D. Homicide, suicide, and unintentional firearm mortality: comparing the United States with other high-income countries, 2003. *Journal of Trauma* 2011; 70:238-243.

⁶ Centers for Disease Control and Prevention. Web-based Injury Statistics Query and Reporting System (WISQARS) [Online]. National Center for Injury Control and Prevention, Centers for Disease Control and Prevention (producer). Available from: URL: <http://www.cdc.gov/injury/wisqars/index.html>. [2012, Mar. 22].

⁷ Cook PJ, Ludwig J. Gun Violence: The Real Costs. New York: Oxford University Press, 2000.

⁸ Shapiro RJ, Hassett KA. *The Economic Benefits of Reducing Violent Crime: A Case Study of 8 American Cities*. Center for American Progress, Washington, DC, June 2012.

⁹ 18 U.S.C. § 922(d) (2012).

¹⁰ Campbell JC, Webster DW, Koziol-McLain J, et al. Risk factors for femicide within physically abusive intimate relationships: Results from a multi-site case control study. *American Journal of Public Health* 2003; 93:1089-1097.

¹¹ Etter, GW, Birzer, ML. Domestic violence abusers: A descriptive study of the characteristics of defenders in protection form abuse orders in Sedgwick County, Kansas. *Journal of Family Violence*. 2007; 22:113-119.

¹² Fagan JA, Stewart DK, Hansen KV. Violent Men or Violent Husbands? Background Factors and Situational Correlates. In D. Finkelhor, R. Gelles, G. Hotaling, M.A. Straus (Eds.), *The Dark Side of Families: Current Family Violence Research* (pp. 49-68). Beverly Hills, CA: Sage Publications, 1983.

¹³ Gayford JJ. Wife battering: A preliminary study of 100 cases. *British Medical Journal*. 1975; 1: 194-197.

¹⁴ Hotaling GT, Straus MA, Lincoln, AJ. Intrafamily Violence, and Crime and Violence Outside the Family. In L. Ohlin, M. Tonry (Eds.) *Family Violence* (pp. 315-375). Chicago: University of Chicago Press, 1989.

¹⁵ Sharps PW, Campbell JC, Campbell D, et al. The role of alcohol use in intimate partner femicide. *American Journal of Addiction*. 2001;10:122-35.

¹⁶ Rivara FP, Mueller BA, Somes G, et al. Alcohol and illicit drug abuse and the risk of violent death in the home. *JAMA*. 1997;278:569-75.

¹⁷ Kelleher K, Chaffin M, Hollenberg J, et al. Alcohol and drug disorders among physically abusive and neglectful parents in a community-based sample. *American Journal of Public Health*. 1994;84:1586-90.

¹⁸ Walton-Moss BJ, Manganello J, Frye V, et al. Risk factors for intimate partner violence and associated injury among urban women. *Journal of Community Health*. 2005;30:377-89.

¹⁹ McClelland GM, Teplin LA. Alcohol intoxication and violent crime: implications for public health policy. *American Journal of Addiction*. 2001;10(Suppl):70-85.

²⁰ Rivara FP, Mueller BA, Somes G, et al. Alcohol and illicit drug abuse and the risk of violent death in the home. *JAMA*. 1997;278:569-75.

²¹ Borowsky IW, Ireland M, Resnick MD. Adolescent suicide attempts: risks and protectors. *Pediatrics*. 2001;107:485-93.

²² Rivara FP, Mueller BA, Somes G, et al. Alcohol and illicit drug abuse and the risk of violent death in the home. *JAMA*. 1997;278:569-75.

²³ Swanson JW, Swartz MS, Van Dorn RA, Elbogen EB, Wagner HR, Rosenheck RA, et al. A National Study of Violent Behavior in Persons With Schizophrenia. *Archives of General Psychiatry*. 2006;63(5):490-9.

²⁴ Swanson J. Mental disorder, substance abuse, and community violence: An epidemiological approach. In: Monahan J, Steadman JH, editors. *Violence and Mental Disorders: Developments in Risk Assessment*. Chicago: University of Chicago Press; 1994.

²⁵ Swanson J. Mental disorder, substance abuse, and community violence: An epidemiological approach. In: Monahan J, Steadman JH, editors. *Violence and Mental Disorders: Developments in Risk Assessment* Chicago: University of Chicago Press; 1994.

²⁶ Monahan J, Steadman H, Silver E, Appelbaum P, Robbins P, Mulvey E, et al. *Rethinking Risk Assessment: The MacArthur Study of Mental Disorder and Violence*. New York: Oxford University Press. 2001.

²⁷ Swanson J, Borum R, Swartz M, et al. Violent behavior preceding hospitalization among persons with severe mental illness. *Social Psychiatry and Psychiatric Epidemiology*. 1999;33:75-80.

²⁸ Nock MK, Borges G, Bromet EJ, Cha CB, Kessler RC, Lee S. Suicide and Suicidal Behavior. *Epidemiologic Reviews*. 2008; 30, 133-154.

-
- ²⁹ Fox JA, Zawitz MW. Homicide trends in the US: age, gender, and race trends. Bureau of Justice Statistics, US Department of Justice, Washington DC. 2010.
- ³⁰ Fabio A, Loeber R, Balasubramani GK, Roth J, Fu W, Farrington DP. Why some generations are more violent than others: assessment of age, period, and cohort effects. *Am J Epidemiology*. 2006;164(2):151-60.
- ³¹ Steinberg L. Risk taking in adolescence: what changes, and why? . *Ann N Y Acad Sci*. 2004;1021:51-8.
- ³² Johnson SB, Blum RW, Giedd JN. Adolescent Maturity and the Brain: The Promise and Pitfalls of Neuroscience Research in Adolescent Health Policy. *Journal of Adolescent Health*. 2009;45(3):216-21.
- ³³ Vittes KA, Vernick JS, Webster DW. Legal status and source of offenders' firearms in states with the least stringent criteria for gun ownership. *Injury Prevention* 2012; Epub.
- ³⁴ Wintemute GJ, Drake CM, Beaumont JJ, Wright MA. Prior misdemeanor convictions as a risk factor for later violent and firearm-related criminal activity among authorized purchasers of handguns. *JAMA*. 1998;280:2083-7.
- ³⁵ Wright MA, Wintemute GJ. Felonious or violent criminal activity that prohibits gun ownership among prior handgun purchasers: incidence and risk factors. *J Trauma* 2010;69:948-55.
- ³⁶ 18 U.S.C § 922(g)(3) (2012); 27 C.F.R. § 478.11 (2012).
- ³⁷ Webster DW, Vernick JS. Keeping Firearms from Drug and Alcohol Abusers. *Injury Prevention* 2009;15:425-427.
- ³⁸ Bureau of Justice Statistics. Homicide Trends in the U.S. 2012 [cited 2012 March 20]; Available from: <http://bjs.ojp.usdoj.gov/content/homicide/teens.cfm>.
- ³⁹ O'Malley PM, Wagenaar AC. Effects of minimum drinking age laws on alcohol use, related behaviors and traffic crash involvement among American youth: 1976-1987. *Journal of Studies on Alcohol*. 1991;52:478-491.
- ⁴⁰ Bureau of Justice Statistics. *Background Checks for Firearms Transfer, 2009 – Statistical Table*. Washington, DC: U.S. Department of Justice; 2010.
- ⁴¹ Ludwig J, Cook PJ. The impact of the Brady Act on homicide and suicide rates. *JAMA*. 2000; 284:2718-21.
- ⁴² Vernick JS, Rutkow L, Salmon DA. Availability of litigation as a public health tool for firearm injury prevention: comparison of guns, vaccines, and motor vehicles. *American Journal of Public Health*. 2007;97:1991-1997.

EXHIBIT 4

EXCERPT



Ex. 4

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Right-to-Carry Laws and Violent Crime: A Comprehensive Assessment Using Panel Data and a State-Level Synthetic Control Analysis

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This article uses more complete state panel data (through 2014) and new statistical techniques to estimate the impact on violent crime when states adopt right-to-carry (RTC) concealed handgun laws. Our preferred panel data regression specification, unlike the statistical model of Lott and Mustard that had previously been offered as evidence of crime-reducing RTC laws, both satisfies the parallel trends assumption and generates statistically significant estimates showing RTC laws *increase* overall violent crime. Our synthetic control approach also finds that RTC laws are associated with 13–15 percent *higher* aggregate violent crime rates 10 years after adoption. Using a consensus estimate of the elasticity of crime with respect to incarceration of 0.15, the average RTC state would need to roughly double its prison population to offset the increase in violent crime caused by RTC adoption.

I. INTRODUCTION

For two decades, there has been a spirited academic debate over whether “shall-issue” concealed carry laws (also known as right-to-carry or RTC laws) have an important impact on crime. The “More Guns, Less Crime” hypothesis originally articulated by John Lott and David Mustard (1997) claimed that RTC laws decreased violent

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crime (possibly shifting criminals in the direction of committing more property crime to avoid armed citizens). This research may well have encouraged state legislatures to adopt RTC laws, arguably making the pair's 1997 paper in the *Journal of Legal Studies* one of the most consequential criminological articles published in the last 25 years.

The original Lott and Mustard paper as well as subsequent work by John Lott in his 1998 book *More Guns, Less Crime* used a panel data analysis to support the theory that RTC laws reduce violent crime. A large number of papers examined the Lott thesis, with decidedly mixed results. An array of studies, primarily those using the limited data initially employed by Lott and Mustard for the period 1977–1992 and those failing to adjust their standard errors by clustering, supported the Lott and Mustard thesis, while a host of other papers were skeptical of the Lott findings.¹

It was hoped that the 2005 National Research Council report *Firearms and Violence: A Critical Review* (hereafter the NRC Report) would resolve the controversy over the impact of RTC laws, but this was not to be. While one member of the committee—James Q. Wilson—did partially endorse the Lott thesis by saying there was evidence that murders fell when RTC laws were adopted, the other 15 members of the panel pointedly criticized Wilson's claim, saying that “the scientific evidence does not support his position.” The majority emphasized that the estimated effects of RTC laws were highly sensitive to the particular choice of explanatory variables and thus concluded that the panel data evidence through 2000 was too fragile to support any conclusion about the true effects of these laws.

This article answers the call of the NRC Report for more and better data and new statistical techniques to be brought to bear on the issue of the impact of RTC laws on crime. First, we revisit the state panel data evidence to see if extending the data for an additional 14 years, thereby providing additional crime data for prior RTC states as well as on 11 newly adopting RTC states, offers any clearer picture of the causal impact of allowing citizens to carry concealed weapons. We distill from an array of different panel data regressions for various crime categories for two time periods using two major sets of explanatory variables—including our preferred specification (DAW) and that of Lott and Mustard (LM)—a subset of regressions that satisfy the critical parallel trends assumption. All the statistically significant results from these regressions show RTC laws are associated with *higher* rates of overall violent crime, property crime, or murder.

Second, to address some of the weaknesses of panel data models, we undertake an extensive synthetic control analysis in order to present the most complete and robust

¹In support of Lott and Mustard (1997), see Lott's 1998 book *More Guns, Less Crime* (and the 2000 and 2010 editions). Ayres and Donohue (2003) and the 2005 National Research Council report *Firearms and Violence: A Critical Review* dismissed the Lott/Mustard hypothesis as lacking credible statistical support, as did Aneja et al. (2011) (and Aneja et al. (2014) further expanding the latter). Moody and Marvell (2008) and Moody et al. (2014) continued to argue in favor of a crime-reducing effect of RTC laws, although Zimmerman (2014) and McElroy and Wang (2017) find that RTC laws *increase* violent crime and Siegel et al. (2017) find RTC laws increase murders, as discussed in Section III.B.

results to guide policy in this area.² This synthetic control methodology—first introduced in Abadie and Gardeazabal (2003) and expanded in Abadie et al. (2010, 2014)—uses a matching methodology to create a credible “synthetic control” based on a weighted average of other states that best matches the prepassage pattern of crime for each “treated” state, which can then be used to estimate the likely path of crime if RTC-adopting states had not adopted an RTC law. By comparing the actual crime pattern for RTC-adopting states with the estimated synthetic controls in the postpassage period, we derive year-by-year estimates for the impact of RTC laws in the 10 years following adoption.³

To preview our major findings, the synthetic control estimate of the average impact of RTC laws across the 33 states that adopt between 1981 and 2007⁴ indicates that violent crime is substantially higher after 10 years than would have been the case had the RTC law not been adopted. Essentially, for violent crime, the synthetic control approach provides a similar portrayal of RTC laws as that provided by the DAW panel data model and undermines the results of the LM panel data model. According to the aggregate synthetic control models—regardless of whether one uses the DAW or LM covariates—RTC laws led to increases in violent crime of 13–15 percent after 10 years, with positive but not statistically significant effects on property crime and murder. The median effect of RTC adoption after 10 years is 12.3 percent if one considers all 31 states with 10 years worth of data and 11.1 percent if one limits the analysis to the 26 states with the most compelling prepassage fit between the adopting states and their synthetic controls. Comparing our DAW specification findings with the results generated using placebo treatments, we are able to reject the null hypothesis that RTC laws have no impact on aggregate violent crime.

The structure of the article proceeds as follows. Section II begins with a discussion of the ways in which increased carrying of guns could either dampen crime (by thwarting or deterring criminals) or increase crime by directly facilitating violence or aggression by permit holders (or others), greatly expanding the loss and theft of guns, and burdening the functioning of the police in ways that diminish their effectiveness in controlling crime. We then show that a simple comparison of the drop in violent crime from

²Abadie et al. (2014) identify a number of possible problems with panel regression techniques, including the danger of extrapolation when the observable characteristics of the treated area are outside the range of the corresponding characteristics for the other observations in the sample.

³The accuracy of this matching can be qualitatively assessed by examining the root mean square prediction error (RMSPE) of the synthetic control in the pretreatment period (or a variation on this RMSPE implemented in this article), and the statistical significance of the estimated treatment effect can be approximated by running a series of placebo estimates and examining the size of the estimated treatment effect in comparison to the distribution of placebo treatment effects.

⁴Note that we do not supply a synthetic control estimate for Indiana, even though it passed its RTC law in 1980, owing to the fact that we do not have enough pretreatment years to accurately match the state with an appropriate synthetic control. Including Indiana as a treatment state, though, would not meaningfully change our results. Similarly, we do not generate synthetic control estimates for Iowa and Wisconsin (whose RTC laws went into effect in 2011) or for Illinois (2014 RTC law), because of the limited postpassage data.

1977–2014 in the states that have resisted the adoption of RTC laws is almost an order of magnitude greater than in RTC-adopting states (a 42.3 percent drop vs. a 4.3 percent drop), although a spartan panel data model with only state and year effects reduces the differential to 20.2 percent. Section III discusses the panel data results, showing that the DAW model indicates that RTC laws have increased violent and property crime, with weaker evidence that RTC laws increased homicide (but not non-gun homicide) over our entire data period, while both the DAW and the LM model provide statistically significant evidence that RTC laws have increased murder in the postcrack period.

The remainder of the article shows that, using either the DAW or LM explanatory variables, the synthetic control approach uniformly supports the conclusion that RTC laws lead to substantial increases in violent crime. Section IV describes the details of our implementation of the synthetic control approach and shows that the mean and median estimates of the impact of RTC laws show greater than double-digit increases by the 10th year after adoption. Section V provides aggregate synthetic control estimates of the impact of RTC laws, and Section VI concludes.

II. THE IMPACT OF RTC LAWS: THEORETICAL CONSIDERATIONS AND SIMPLE COMPARISONS

A. *Gun Carrying and Crime*

1. Mechanisms of Crime Reduction

Allowing citizens to carry concealed handguns can influence violent crime in a number of ways, some benign and some invidious. Violent crime can fall if criminals are deterred by the prospect of meeting armed resistance, and potential victims or armed bystanders may thwart or terminate attacks by either brandishing weapons or actually firing on the potential assailants. For example, in 2012, a Pennsylvania concealed carry permit holder became angry when he was asked to leave a bar because he was carrying a weapon and, in the ensuing argument, he shot two men, killing one, before another permit holder shot him (Kalinowski 2012). Two years later, a psychiatric patient in Pennsylvania killed his caseworker, and grazed his psychiatrist before the doctor shot back with his own gun, ending the assault by wounding the assailant (Associated Press 2014).

The impact of the Pennsylvania RTC law is somewhat ambiguous in both these cases. In the bar shooting, it was a permit holder who started the killing and another who ended it, so the RTC law may actually have increased crime. The case of the doctor's use of force is more clearly benign, although the RTC law may have made no difference: a doctor who routinely deals with violent and deranged patients would typically be able to secure a permit to carry a gun even under a may-issue regime. Only a statistical analysis can reveal whether in aggregate extending gun carrying beyond those with a demonstrated need and good character, as shall-issue laws do, imposes or reduces overall costs.

Some defensive gun uses can be socially costly and contentious even if they do avoid a robbery or an assault. For example, in 1984, when four teens accosted Bernie Goetz on a New York City subway, he prevented an anticipated robbery by shooting all four,

Everything that takes up added police time or complicates the job of law enforcement will serve as a tax on police, rendering them less effective on the margin, and thereby contributing to crime. Indeed, this may in part explain why RTC states tend to increase the size of their police forces (relative to nonadopting states) after RTC laws are passed, as shown in Table 1.³³

B. A Simple Difference-in-Differences Analysis

We begin by showing how violent crime evolved over our 1977–2014 data period for RTC and non-RTC states.³⁴ Figure 1 depicts percentage changes in the violent crime rate over our entire data period for three groups of states: those that never adopted RTC laws, those that adopted RTC laws sometime between 1977 and before 2014, and those that adopted RTC laws prior to 1977. It is noteworthy that the 42.3 percent drop in violent crime in the nine states that never adopted RTC laws is almost an order of magnitude greater than the 4.3 percent reduction experienced by states that adopted RTC laws during our period of analysis.³⁵

The NRC Report presented a “no-controls” estimate, which is just the coefficient estimate on the variable indicating the date of adoption of a RTC law in a crime rate panel data model with state and year fixed effects. According to the NRC Report: “Estimating the model using data to 2000 shows that states adopting right-to-carry laws saw 12.9 percent increases in violent crime—and 21.2 percent increases in property crime—relative to national crime patterns.” Estimating this same model using 14 additional years of data (through 2014) and 11 additional adopting states (listed at the bottom of Appendix Table C1) reveals that the average postpassage increase in violent crime was

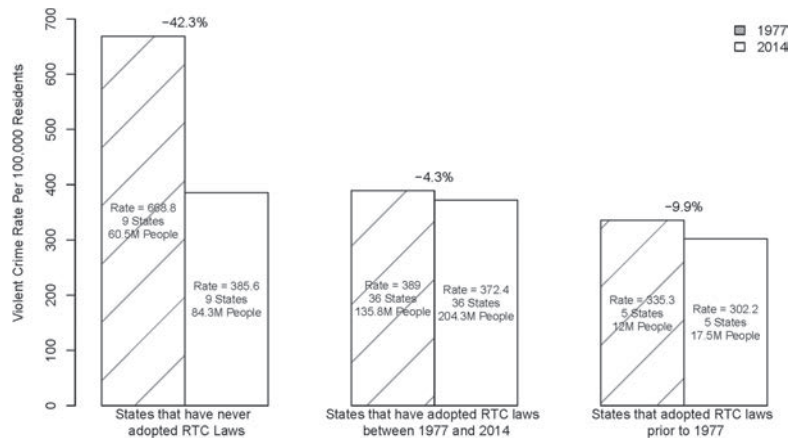
Indiana was shot by her three-year-old daughter when the toddler’s father left the legal but loaded 9mm handgun between the console and the front passenger seat after he exited the vehicle to go inside a store. The child climbed over from the backseat and accidentally fired the gun, hitting her mother through the upper right part of her torso. (Palmer 2018) See also Savitsky (2019) (country western singer Justin Carter dies when the gun in his pocket discharges and hits him in the face); Schwarz (2014) (Idaho professor shoots himself in foot during class two months after state legalizes guns on campuses); Murdock (2018) (man shoots himself in the groin with gun in his waistband in the meat section of Walmart in Buckeye, Arizona); Barbash (2018) (California teacher demonstrating gun safety accidentally discharges weapon in a high school classroom in March 2018, injuring one student); Fortin (2018) (in February 2018, a Georgia teacher fired his gun while barricaded in his classroom); US News (2018) (in April 2018, an Ohio woman with a valid concealed carry permit accidentally killed her two-year-old daughter at an Ohio hotel while trying to turn on the gun’s safety); and Fox News (2016) (“the owner of an Ohio gun shop was shot and killed when a student in a concealed carry permit class accidentally discharged a weapon,” striking the owner in the neck in a different room after the bullet passed through a wall).

³³See Adda et al. (2014), describing how local depenalization of cannabis enabled the police to reallocate resources, thereby reducing violent crime.

³⁴The FBI violent crime category includes murder, rape, robbery, and aggravated assault.

³⁵Over the same 1977–2014 period, the states that avoided adopting RTC laws had substantially smaller increases in their rates of incarceration and police employment. The nine never-adopting states increased their incarceration rate by 205 percent, while the incarceration rates in the adopting states rose by 262 and 259 percent, for those adopting RTC laws before and after 1977, respectively. Similarly, the rate of police employment rose by 16 percent in the never-adopting states and by 38 and 55 percent for those adopting before and after 1977, respectively.

Figure 1: The decline in violent crime rates has been far greater in states with no RTC laws, 1977–2014.



DATA SOURCES: UCR for crime rates; Census for state populations.

NOTE: Illinois excluded since its concealed carry law did not go into effect until 2014. From 1977–2013, the violent crime rate in Illinois fell by 36 percent, from 631 to 403 crimes per 100,000 people.

20.2 percent, while the comparable increase in property crime was 19.2 percent (both having *p* values less than 5 percent).³⁶

Of course, it does not prove that RTC laws increase crime simply because RTC states experience a worse postpassage crime pattern. For example, it might be the case that some states decided to fight crime by allowing citizens to carry concealed handguns while others decided to hire more police and incarcerate a greater number of convicted criminals. If police and prisons were more effective in stopping crime, the “no-controls” model might show that the crime experience in RTC states was worse than in other states even if this were not a true causal result of the adoption of RTC laws. As it turns out, though, RTC states not only experienced higher rates of violent crime but they also had larger increases in incarceration and police than other states. Table 1 provides panel data evidence on how incarceration and two measures of police employment changed after RTC adoption (relative to nonadopting states). All three measures rose in RTC states, and the 7–8 percent greater increases in police in RTC states are statistically significant. In other words, Table 1 confirms that RTC states did *not* have relatively declining rates of

³⁶The dummy variable model reports the coefficient associated with a RTC variable that is given a value of 0 when a RTC law is not in effect in that year, a value of 1 when a RTC law is in effect that entire year, and a value equal to the portion of the year a RTC law is in effect otherwise. The date of adoption for each RTC state is shown in Appendix Table A1. Note the fact that violent crime was noticeably higher in 1977 in the nine states that did not adopt RTC laws indicates that it will be particularly important that the parallel trends requirement of a valid panel data analysis is established, which is an issue to which we carefully attend in Section III.A.3. All our appendices are posted online at https://works.bepress.com/john_donohue/.

Table 1: Panel Data Estimates Showing Greater Increases in Incarceration and Police Following RTC Adoption: State- and Year-Fixed Effects, and No Other Regressors, 1977–2014

	<i>Incarceration</i>	<i>Police Employment per 100k</i>	<i>Police Officers per 100k</i>
	(1)	(2)	(3)
Dummy variable model	6.78 (6.22)	8.39*** (3.15)	7.08** (2.76)

NOTE: OLS estimations include state- and year-fixed effects and are weighted by population. Robust standard errors (clustered at the state level) are provided next to point estimates in parentheses. The police employment and sworn police officer data are from the Uniform Crime Reports (UCR). The source of the incarceration rate is the Bureau of Justice Statistics (2014). * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$. All figures reported in percentage terms.

incarceration or total police employees after adopting their RTC laws that might explain their comparatively poor postpassage crime performance.

III. A PANEL DATA ANALYSIS OF RTC LAWS

A. Estimating Two Models on the Full Data Period 1977–2014

We have just seen that RTC law adoption is followed by *higher* rates of violent and property crime (relative to national trends) and that the elevated crime levels after RTC law adoption occur despite the fact that RTC states actually invested relatively more heavily in prisons and police than non-RTC states. While the theoretical predictions about the effect of RTC laws on crime are indeterminate, these two empirical facts based on the actual patterns of crime and crime-fighting measures in RTC and non-RTC states suggest that the most plausible working hypothesis is that RTC laws *increase* crime. The next step in a panel data analysis of RTC laws would be to test this hypothesis by introducing an appropriate set of explanatory variables that plausibly influence crime.

The choice of these variables is important because any variable that both influences crime and is simultaneously correlated with RTC laws must be included if we are to generate unbiased estimates of the impact of RTC laws. At the same time, including irrelevant and/or highly collinear variables can also undermine efforts at valid estimation of the impact of RTC laws. At the very least, it seems advisable to control for the levels of police and incarceration because these have been the two most important criminal justice policy instruments in the battle against crime.

1. The DAW Panel Data Model

In addition to the state and year fixed effects of the no-controls model and the identifier for the presence of an RTC law, our preferred “DAW model” includes an array of other factors that might be expected to influence crime, such as the levels of police and incarceration, various income, poverty, and unemployment measures, and six demographic controls designed to capture the presence of males in three racial categories (black, white, other) in two high-crime age groupings (15–19 and 20–39). Table 2 lists the full

Table 2: Table of Explanatory Variables for Four Panel Data Studies

<i>Explanatory Variables</i>	<i>DAW</i>	<i>LM</i>
Right-to-carry law	x	x
Lagged per capita incarceration rate	x	
Lagged police staffing per 100,000 residents	x	
Poverty rate	x	
Unemployment rate	x	
Per capita ethanol consumption from beer	x	
Percentage of state population living in metropolitan statistical areas (MSA)	x	
Real per capita personal income	x	x
Real per capita income maintenance		x
Real per capita retirement payments		x
Real per capita unemployment insurance payments		x
Population density		x
Lagged violent or property arrest rate		x
State population		x
6 Age-sex-race demographic variables —all 6 combinations of black, white, and other males in 2 age groups (15–19, 20–39) indicating the percentage of the population in each group	x	
36 Age-sex-race demographic variables —all possible combinations of black, white, and other males in 6 age groups (10–19, 20–29, 30–39, 40–49, 50–64, and over 65) and repeating this all for females, indicating the percentage of the population in each group		x

NOTE: The DAW model is advanced in this article and the LM model was previously published by Lott and Mustard.

set of explanatory variables for both the DAW model and the comparable panel data model used by Lott and Mustard (LM).³⁷

Mathematically, the simple dummy model takes the following form:

$$\ln(\text{crime rate}_{it}) = \beta X_{it} + \gamma RTC_{it} + \alpha_t + \delta_i + \varepsilon_{it} \quad (1)$$

where γ is the coefficient on the RTC dummy, reflecting the average estimated impact of adopting a RTC law on crime. The matrix X_{it} contains either the DAW or LM covariates

³⁷While we attempt to include as many state-year observations in these regressions as possible, District of Columbia incarceration data are missing after the year 2001. In addition, a handful of observations are also dropped from the LM regressions owing to states that did not report any usable arrest data in various years. Our regressions are performed with Huber-White robust standard errors that are clustered at the state level, and we lag the arrest rates used in the LM regression models. The rationales underlying both choices are described in more detail in Aneja et al. (2014). All the regressions presented in this article are weighted by state population.

new techniques should be employed to estimate the impact of these laws is fitting. The important paper by Starnad (2007) used a Bayesian approach to argue that none of the published models used in the RTC evaluation literature rated highly in his model selection protocol when applied to data from 1977–1999.

Durlauf et al. attempt to sort out the different specification choices in evaluating RTC laws by using their own Bayesian model averaging approach using county data from 1979–2000. Applying this technique, the authors find that in their preferred spline (trend) model, RTC laws elevate violent crime in the three years after RTC adoption: “As a result of the law being introduced, violent crime increases in the first year and continues to increase afterwards” (2016:50). By the third year, their preferred model suggests a 6.5 percent increase in violent crime. Since their paper only provides estimates for three postpassage years, we cannot draw conclusions beyond this but note that their finding that violent crime increases by over 2 percent per year owing to RTC laws is a substantial crime increase. Moreover, the authors note: “For our estimates, the effect on crime of introducing guns continues to grow over time” (2016:50).⁴⁶

Owing to the substantial challenges of estimating effects from observational data, it will be useful to see if yet another statistical approach that has different attributes from the panel data methodology can enhance our understanding of the impact of RTC laws. The rest of this article will use this synthetic control approach, which has been deemed “arguably the most important innovation in the policy evaluation literature in the last 15 years” (Athey & Imbens 2017).

IV. ESTIMATING THE IMPACT OF RTC LAWS USING SYNTHETIC CONTROLS

The synthetic control methodology, which is becoming increasingly prominent in economics and other social sciences, is a promising new statistical approach for addressing the impact of RTC laws.⁴⁷ While most synthetic control papers focus on a single

⁴⁶While our analysis focused on crime at the state level, there is obviously heterogeneity in crime rates within states, which is amalgamated into our population-weighted state average figures. A paper by Kovandzic et al. (KMV) buttresses the view that our state-focused estimates are not giving a misleading impression of the impact of RTC laws on violent crime. KMV limited their analysis to urban areas within each state, estimating the impact of RTC laws on crime using a panel data analysis from 1980–2000 on 189 cities with a population of 100,000 or more (Kovandzic et al. 2005). Although they did not estimate an overall violent crime effect, they did report that RTC laws were associated with a highly statistically significant increase in the rate of aggravated assault, the largest single component of violent crime. Their figures suggest that RTC laws led to a 20.1 percent increase in aggravated assault in the 10 years following adoption.

⁴⁷The synthetic control methodology has been deployed in a wide variety of fields, including health economics (Nonnemaker et al. 2011), immigration economics (Bohn et al. 2014), political economy (Keele 2009), urban economics (Ando 2015), the economics of natural resources (Mideksa 2013), and the dynamics of economic growth (Cavallo et al. 2013).

treatment in a single geographic region, we look at 33 RTC adoptions occurring over three decades throughout the country. For each adopting (“treated”) state we will find a weighted average of other states (“a synthetic control”) designed to serve as a good counterfactual for the impact of RTC laws because it had a pattern of crime similar to that of the adopting state prior to RTC adoption. By comparing what actually happened to crime after RTC adoption to the crime performance of the synthetic control over the same period, we generate estimates of the causal impact of RTC laws on crime.⁴⁸

A. The Basics of the Synthetic Control Methodology

The synthetic control method attempts to generate representative counterfactual units by comparing a treatment unit (i.e., a state adopting an RTC law) to a set of control units across a set of explanatory variables over a preintervention period. The algorithm searches for similarities between the treatment state of interest and the control states during this period and then generates a synthetic counterfactual unit for the treatment state that is a weighted combination of the component control states.⁴⁹ Two conditions are placed on these weights: they must be nonnegative and they must sum to 1. In general, the matching process underlying the synthetic control technique uses pretreatment values of both the outcome variable of interest (in our case, some measure of crime) and other predictors believed to influence this outcome variable.⁵⁰ For the reasons set forth in Appendix K, we use every lag of the dependent variable as predictors in the DAW and LM specifications. Once the synthetic counterfactual is generated and the weights associated with each control unit are assigned, the *synth* program then calculates values for the outcome variable associated with this counterfactual and the root mean squared prediction error (RMSPE) based on differences between the treatment and synthetic control units in the pretreatment period. The effect of the treatment can then be estimated by comparing the actual values of the dependent variable for the treatment unit to the corresponding values of the synthetic control.

B. Generating Synthetic Controls for 33 States Adopting RTC Laws During Our Data Period

To illustrate the procedure outlined above, consider the case of Texas, whose RTC law went into effect on January 1, 1996. The potential control group for each treatment state

⁴⁸For a more detailed technical description of this method, we direct the reader to Abadie and Gardeazabal (2003) and Abadie et al. (2010, 2014).

⁴⁹Our analysis is done in Stata using the *synth* software package developed by Alberto Abadie, Alexis Diamond, and Jens Hainmueller.

⁵⁰Roughly speaking, the algorithm that we use finds \mathbf{W} (the weights of the components of the synthetic control) that minimizes $\sqrt{(\mathbf{X}_1 - \mathbf{X}_0\mathbf{W})' \mathbf{V}(\mathbf{X}_1 - \mathbf{X}_0\mathbf{W})}$, where \mathbf{V} is a diagonal matrix incorporating information about the relative weights placed on different predictors, \mathbf{W} is a vector of nonnegative weights that sum to 1, \mathbf{X}_1 is a vector containing pretreatment information about the predictors associated with the treatment unit, and \mathbf{X}_0 is a matrix containing pretreatment information about the predictors for all the control units.

consists of all nine states with no RTC legislation as of the year 2014, as well as states that pass RTC laws at least 10 years after the passage of the treatment state (e.g., in this case, the five states passing RTC laws after 2006, such as Nebraska and Kansas, whose RTC laws went into effect at the beginning of 2007). Since we estimate results for up to 10 years postpassage,⁵¹ this restriction helps us avoid including states with their own permissive concealed carry laws in the synthetically constructed unit (which would mar the control comparison).

After entering the necessary specification information into the *synth* program (e.g., treatment unit, list of control states, explanatory variables, etc.), the algorithm proceeds to construct the synthetic unit from the list of control states specific to Texas and generates values of the dependent variable for the counterfactual for both the pre-treatment and posttreatment periods. The rationale behind this methodology is that a close fit in the prepassage time series of crime between the treatment state and the synthetic control generates greater confidence in the accuracy of the constructed counterfactual. Computing the posttreatment difference between the dependent variables of the treatment state and the synthetic control unit provides the synthetic control estimate of the treatment effect attributable to RTC adoption in that state.

1. Synthetic Control Estimates of Violent Crime in Two States

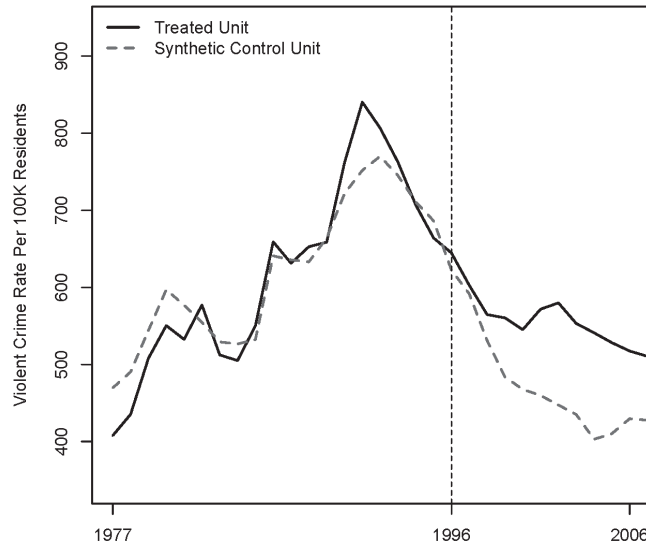
Figure 5 shows the synthetic control graph for violent crime in Texas over the period from 1977 through 2006 (10 years after the adoption of Texas's RTC law). The solid black line shows the actual pattern of violent crime for Texas, and the vertical line indicates when the RTC law went into effect. Implementing the synthetic control protocol identifies three states that generate a good fit for the pattern of crime experienced by Texas in the pre-1996 period. These states are California, which gets a weight of 57.7 percent owing to its similar attributes compared to Texas, Nebraska with a weight of 9.7 percent, and Wisconsin with a weight of 32.6 percent.

One of the advantages of the synthetic control methodology is that one can assess how well the synthetic control (call it "synthetic Texas," which is identified in Figure 5 by the dashed line) matches the pre-RTC-passage pattern of violent crime to see whether the methodology is likely to generate a good fit in the 10 years of postpassage data. Here the fit looks rather good in mimicking the rises and falls in Texas violent crime from 1977–1995. This pattern increases our confidence that synthetic Texas will provide a good prediction of what would have happened in Texas had it not adopted an RTC law.

Looking at Figure 5, we see that while both Texas and synthetic Texas (the weighted average violent crime performance of the three mentioned states) show declining crime rates in the postpassage decade after 1996, the crime drop is

⁵¹Our choice of 10 years is informed by the tradeoffs associated with using a different timeframe. Tables 5 and 6 indicate that the increase in violent crime due to RTC laws is statistically significant at the .01 level for all years after seven years post-adoption.

Figure 5: Texas: Violent crime rate.



Effect of 1996 RTC Law 10 Years After Adoption: 16.9%

NOTE: Passage Year Difference From SC: 3.6% Composition of SC: CA (0.577); NE (0.097); WI (0.326) CVRMSPE: 0.06 (8 of 33 states, where 1 denotes the state with the best pre-passage fit.).

States Never Passing RTC Laws Included in Synthetic Control: CA;

RTC Adopting States Included in Synthetic Control: NE (2007); WI (2012).

substantially greater in synthetic Texas, which had no RTC law over that period, than in actual Texas, which did. As Figure 5 notes, 10 years after adopting its RTC law, violent crime in Texas was 16.9 percent *higher* than we would have expected had it not adopted an RTC law.⁵²

Figure 5 also illustrates perhaps the most important lesson of causal inference: one cannot simply look before and after an event to determine the consequence of the event. Rather, one needs to estimate the difference between what did unfold and the counterfactual of what would have unfolded without the event. The value of the synthetic control methodology is that it provides a highly transparent estimate of that counterfactual, using a tool designed to ensure the validity of the parallel trends assumption that we have already seen is so critical to achieving meaningful causal estimates. Thus, when Lott

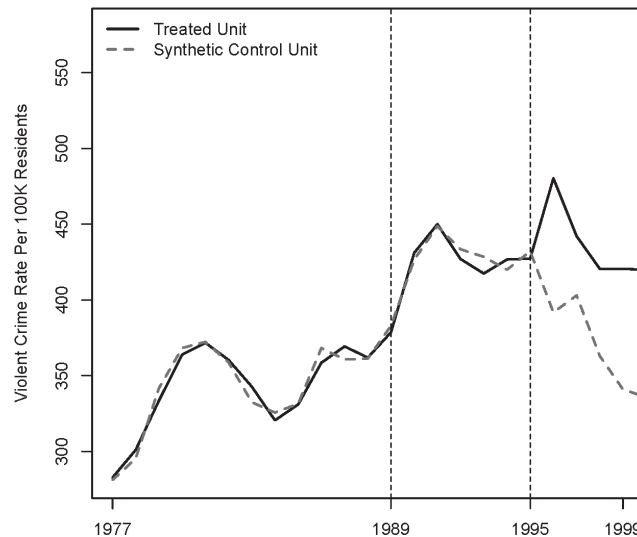
⁵²Texas's violent crime rate 10 years post-adoption exceeds that of "synthetic Texas" by 20.41 percent $= \frac{517.3 - 429.6}{429.6} \times 100\%$. While some researchers would take that value as the estimated effect of RTC, we chose to subtract off the discrepancy in 1996 between the actual violent crime rate and the synthetic control value in that year. This discrepancy is 3.55 percent $= \frac{644.4 - 622.3}{622.3} \times 100\%$ (shown in the line just below the graph of Figure 5). See footnote 58 for further discussion of this calculation. Figure 5 shows a (rounded) estimated violent crime increase in Texas of 16.9 percent. We arrive at this estimate by subtracting the 1996 discrepancy of 3.55 percent from the 20.41 percent 10th-year discrepancy, which generates a TEP of 16.86 percent.

(2010) quotes a Texas District Attorney suggesting that he had reversed his earlier opposition to the state's RTC law in light of the perceived favorable experience with the law, we see why it can be quite easy to draw the inaccurate causal inference that Texas's crime decline was facilitated by its RTC law. The public may perceive the falling crime rate post-1996 (the solid black line), but our analysis suggests that Texas would have experienced a more sizable violent crime decline if it had not passed an RTC law (the dotted line). More specifically, Texas experienced a 19.7 percent decrease in its aggregate violent crime rate in the 10 years following its RTC law (between 1996 and 2006), while the state's synthetic control experienced a larger 31.0 percent decline. This counterfactual would not be apparent to residents of the state or to law enforcement officials, but our results suggest that Texas's RTC law imposed a large social cost on the state.

The greater transparency of the synthetic control approach is one advantage of this methodology over the panel data models that we considered above. Figure 5 makes clear what Texas is being compared to, and we can reflect on whether this match is plausible and whether anything other than RTC laws changed in these three states during the post-passage decade that might compromise the validity of the synthetic control estimate of the impact of RTC laws.

Figure 6 shows our synthetic control estimate for Pennsylvania, which adopted an RTC law in 1989 that did not extend to Philadelphia until a subsequent law went into

Figure 6: Pennsylvania: Violent crime rate.



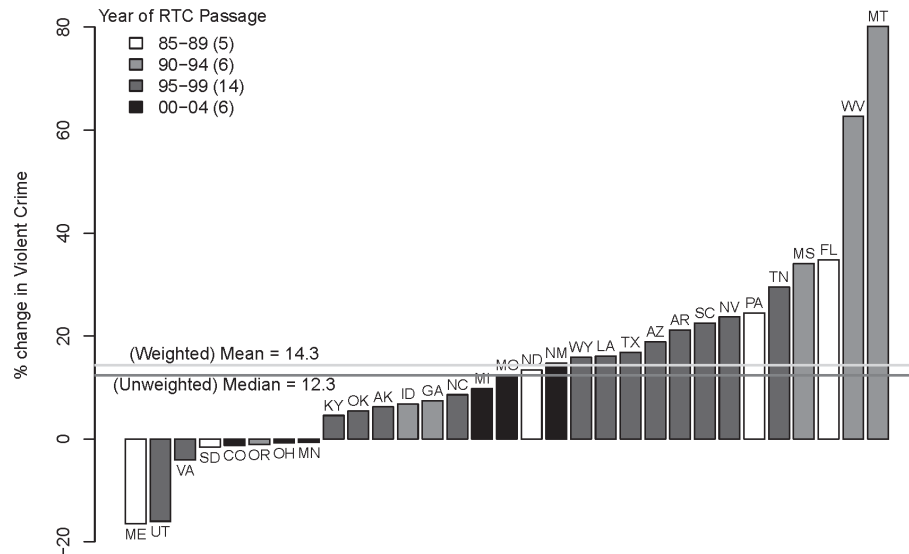
Effect of 1989 RTC Law 10 Years After Adoption: 24.4%

NOTE: Passage Year Difference From SC: -1.1%. Composition of SC: DE (0.078); HI (0.073); MD (0.038); NE (0.016); NJ (0.103); OH (0.27); WI (0.424) CVMSPSE: 0.017 (1 of 33 states, where 1 denotes the state with the best pre-passage fit.).

States Never Passing RTC Laws Included in Synthetic Control: DE; HI; MD; NJ;

RTC Adopting States Included in Synthetic Control: NE (2007); OH (2004); WI (2012).

Figure 7: The effect of RTC laws on violent crime after 10 years, synthetic control estimates for 31 states (1977–2014).



effect on October 11, 1995. In this case, synthetic Pennsylvania is comprised of eight states and the prepassage fit is nearly perfect. Following adoption of the RTC laws, synthetic Pennsylvania shows substantially better crime performance than actual Pennsylvania after the RTC law is extended to Philadelphia in late 1995, as illustrated by the second vertical line at 1996. The synthetic control method estimates that RTC laws in Pennsylvania increased its violent crime rate by 24.4 percent after 10 years.⁵³

2. State-Specific Estimates Across All RTC States

Because we are projecting the violent crime experience of the synthetic control over a 10-year period, there will undoubtedly be a deviation from the “true” counterfactual and our estimated counterfactual. If we were only estimating the impact of a legal change for a single state, we would have an estimate marred by this purely stochastic aspect of changing crime. Since we are estimating an average effect across a large number of states, the

⁵³In Appendix I, we include all 33 graphs showing the path of violent crime for the treatment states and the synthetic controls, along with information about the composition of these synthetic controls, the dates of RTC adoption (if any) for states included in these synthetic controls, and the estimated treatment effect (expressed in terms of the percent change in a particular crime rate) 10 years after adoption (or seven years after adoption for two states that adopted RTC laws in 2007, since our data end in 2014). The figures also document the discrepancy in violent crime in the year of adoption between the actual and synthetic control values.

stochastic variation will be diminished as the overestimates and underestimates will tend to wash out in our mean treatment estimates. Figure 7 shows the synthetic control estimates on violent crime for all 31 states for which we have 10 years of postpassage data. For 23 of the 31 states adopting RTC laws, the increase in violent crime is noteworthy.⁵⁴ Although three states were estimated to have crime reductions greater than the -1.6 percent estimate of South Dakota, if one averages across all 31 states, the (population-weighted) mean treatment effect after 10 years is a 14.3 percent *increase* in violent crime. If one instead uses an (unweighted) median measure of central tendency, RTC laws are seen to *increase* crime by 12.3 percent.

3. Less Effective Prepassage Matches

Section IV.B.1 provided two examples of synthetic controls that matched the crime of the treatment states well in the prepassage period, but this does not always happen. For example, we would have considerably less confidence in the quality of the synthetic control estimates for Maine, whose poor estimate is depicted in Appendix Figure I11. Maine also happens to be the state showing the greatest reduction in violent crime following RTC adoption, as indicated in Figure 7.

For Maine, one sees that the synthetic control and the state violent crime performance diverged long before RTC adoption in 1986, and that, by the date of adoption, Maine's violent crime rate was already 37.9 percent below the synthetic control estimate. The violent crime rate of actual Maine was trending down, while the synthetic control estimate had been much higher and trending up in the immediate pre-adoption period. The difficulty in generating good prepassage matches for states like Maine stems from their unusually low violent crime in the prepassage period.

Appendix Figure D11 reproduces Figure 7 while leaving out the five states for which the quality of prepassage fit is clearly lower than in the remaining 26 states.⁵⁵ This knocks out North Dakota, South Dakota, Maine, Montana, and West Virginia, thereby eliminating three of the five outlier estimates at both ends of the scale, and leaving the mean and median effects of RTC laws relatively unchanged from Figure 7. As Appendix Figure D11 shows, the (weighted) mean increase in crime across the listed 26 RTC-adopting states is 13.7 percent while the (unweighted) median increase is now 11.1 percent. Increases in violent crime of this magnitude are troubling. Consensus estimates of the elasticity of crime with respect to incarceration hover around 0.15 today, which suggests that to offset the increase in crime caused by RTC adoption, the average RTC state would need to approximately double its prison population.

⁵⁴The smallest of these, Kentucky, had an increase of 4.6 percent.

⁵⁵In particular, for these five states, the prepassage CVRMSPE—that is, the RMSPE transformed into a coefficient of variation by dividing by the average prepassage crime rate—was 19 percent or greater. See note 61 for further discussion of this statistic.

V. AGGREGATION ANALYSIS USING SYNTHETIC CONTROLS

A small but growing literature applies synthetic control techniques to the analysis of multiple treatments.⁵⁶ We estimate the percentage difference in violent crime between each treatment (RTC-adopting) state and the corresponding synthetic control in both the year of the treatment and in the 10 years following it. This estimate of the treatment effect percentage (TEP) obviously uses data from fewer posttreatment years for the two treatment states⁵⁷ in which RTC laws took effect less than 10 years before the end of our sample.

We could use each of these 10 percentage differences as our estimated effects of RTC laws on violent crime for the 10 postpassage years, but, as noted above, we make one adjustment to these figures by subtracting from each the percentage difference in violent crime in the adoption year between the treatment and synthetic control states. In other words, if 10 years after adopting an RTC law, the violent crime rate for the state was 440 and the violent crime rate for the synthetic control was 400, one estimate of the effect of the RTC law could be 10 percent ($= \frac{440 - 400}{400}$). Rather than use this estimate, however, we have subtracted from this figure the percentage difference between the synthetic and treatment states in the year of RTC adoption. If, say, the violent crime rate in the treatment state that year was 2 percent higher than the synthetic control value, we would subtract 2 from 10 to obtain an estimated 10th-year effect of RTC laws of 8 percent.⁵⁸ We

⁵⁶The closest paper to the present study is Arindrajit Dube and Ben Zipperer (2013), who introduce their own methodology for aggregating multiple events into a single estimated treatment effect and calculating its significance. Their study centers on the effect of increases in the minimum wage on employment outcomes, and, as we do, the authors estimate the percentage difference between the treatment and the synthetic control in the post-treatment period. While some papers analyze multiple treatments by aggregating the areas affected by these treatments into a single unit, this approach is not well-equipped to deal with a case such as RTC law adoption where treatments affect the majority of panel units and more than two decades separate the dates of the first and last treatment under consideration, as highlighted in Figure 7.

⁵⁷These two states are Kansas and Nebraska, which adopted RTC laws in 2007. See note 4 discussing the states for which we cannot estimate the impact of RTC laws using synthetic controls.

⁵⁸It is unclear *ex ante* whether one should implement this subtraction. The intuitive rationale for our choice of outcome variable was that pretreatment differences between the treatment state and its synthetic control at the time of RTC adoption likely reflected imperfections in the process of generating a synthetic control and should not contribute to our estimated treatment effect if possible. In other words, if the treatment state had a crime rate that was 5 percent greater than that of the synthetic control in both the pretreatment and posttreatment period, it would arguably be misleading to ignore the pretreatment difference and declare that the treatment increased crime rates by 5 percent. On the other hand, subtracting off the initial discrepancy might be adding noise to the subsequent estimates.

We resolve this issue with the following test of our synthetic control protocol: we pretend that each RTC-adopting state actually adopted its RTC law five years before it did. We then generate synthetic control estimates of this phantom law over the next five years of actual pretreatment data. If our synthetic control approach is working perfectly, it should simply replicate the violent crime pattern for the five pretreatment years. Consequently, the estimated “effect” of the phantom law should be close to zero. Indeed, when we follow our subtraction protocol, the synthetic controls match the pretreatment years more closely than when we do not provide this normalization. Specifically, with subtraction the estimated “effect” in the final pretreatment year is a wholly insignificant 3.2 percent; without subtraction, it jumps to a statistically significant 5.3 percent. Consequently,

then look across all the state-specific estimates of the impact of RTC laws on violent crime for each of the 10 individual postpassage years and test whether they are significantly different from zero.⁵⁹

A. RTC Laws Increase Violent Crime

We begin our analysis of the aggregated synthetic control results using predictors derived from the DAW specification. Table 5 shows our results on the full sample examining violent crime.⁶⁰ Our estimates of the normalized average treatment effect percentage (TEP) suggest that states that passed RTC laws experienced more deleterious changes in violent criminal activity than their synthetic controls in the 10 years after adoption. On average, treatment states had aggregate violent crime rates that were almost 7 percent higher than their synthetic controls five years after passage and around 14 percent higher 10 years after passage. Table 5 suggests that the longer the RTC law is in effect (up to the 10th year that we analyze), the greater the cost in terms of increased violent crime.

As we saw in Figures 6 (Pennsylvania) and I11 (Maine), the validity of using the posttreatment difference between crime rates in the treatment state (the particular state adopting an RTC law that we are analyzing) and its corresponding synthetic control as a measure of the effect of the RTC law depends on the strength of the match between these two time series in the pretreatment period. To generate an estimate of pretreatment fit that takes into account differences in pretreatment crime levels, we estimate the coefficient of variation for the root mean squared prediction error (RMSPE), which

normalization is the preferred approach for violent crime. It should also be noted that our actual synthetic control estimates will be expected to perform better than this phantom RTC estimate since we will be able to derive our synthetic controls from five additional years of data, thereby improving our pretreatment fit.

As it turns out, the choice we made to subtract off the initial-year crime discrepancy is a conservative one, in that the estimated crime increases from RTC laws would be *greater* without subtraction. We provide synthetic control estimates for the DAW model without subtraction of the adoption-year percentage difference for violent crime, murder, and property crime in Appendix F. Comparison of these Appendix F estimates with those in the text (Table 5) reveals that our preferred method of subtracting yields more conservative results (i.e., a smaller increase in violent crime due to RTC). In Table 5, we estimate the 10th-year TEP for violent crime as roughly 13.5 to 14.3 percent, while the comparable estimates without subtraction are roughly 17–18 percent, as seen in Appendix Tables F1, F2, and F3. Indeed, without subtraction, every estimated impact would show RTC laws lead to a statistically significant increase in every crime category we consider except non-firearm homicide, as seen in Appendix F.

⁵⁹This test is performed by regressing these differences in a model using only a constant term and examining whether that constant is statistically significant. These regressions are weighted by the population of the treatment state in the posttreatment year under consideration. Robust standard errors corrected for heteroskedasticity are used in this analysis.

⁶⁰We discuss the synthetic control estimates for murder and property crime in Section V.F.

Table 5: The Impact of RTC Laws on the Violent Crime Rate, DAW Covariates, Full Sample, 1977–2014

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Average normalized treatment effect percentage (TEP)	-0.117 (1.076)	2.629* (1.310)	3.631* (1.848)	4.682** (2.068)	6.876*** (2.499)	7.358** (3.135)	10.068*** (2.823)	12.474*** (3.831)	14.021*** (3.605)	14.344*** (2.921)
<i>N</i>	33	33	33	33	33	33	33	31	31	31
Pseudo <i>p</i> value	0.936	0.274	0.220	0.192	0.094	0.106	0.060	0.038	0.032	0.032

NOTE: Standard errors in parentheses. Column numbers indicate postpassage year under consideration; *N* = number of states in sample. The synthetic controls method is run using the nested option, and each year's estimate and statistical significance is computed as explained in note 59. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

is the ratio of the synthetic control's pretreatment RMSPE to the pretreatment average level of the outcome variable for the treatment state.⁶¹

To evaluate the sensitivity of the aggregate synthetic control estimate of the crime impact of RTC laws in Table 5, we consider two subsamples of treatment states: states whose coefficients of variation are less than two times the average coefficient of variation for all 33 treatments and states whose coefficients of variation are less than this average. We then rerun our synthetic control protocol using each of these two subsamples to examine whether restricting our estimation of the average treatment effect to states for which a relatively "better" synthetic control could be identified would meaningfully change our findings.

All three samples yield roughly identical conclusions: RTC laws are consistently shown to increase violent crime, with the 10th-year increase ranging from a low of 13.5 (when we remove the six states with above-average values of the CV RMSPE) to a high of 14.3 percent (Table 5).

B. The Placebo Analysis

Our ability to make valid inferences from our synthetic control estimates depends on the accuracy of our standard error estimation. To test the robustness of the standard errors that we present under the first row of Table 5, we incorporate an analysis using placebo treatment effects similar to Ando (2015).⁶² For this analysis, we generate 500 sets of randomly generated RTC dates that are designed to resemble the distribution of actual RTC

⁶¹While the RMSPE is often used to assess this fit, we believe that the use of this measure is not ideal for comparing fit across states, owing to the wide variation that exists in the average pretreatment crime rates among the 33 treatment states that we consider. For example, the pretreatment RMSPE associated with our synthetic control analysis using the DAW predictor variables and aggregate violent crime as the outcome variable is nearly identical for Texas (37.1) and Maine (36.4), but the pretreatment levels of Texas's aggregate violent crime rate are far greater than Maine's. To be more specific, Texas's average violent crime rate prior to the implementation of its RTC law (from 1977 through 1995) was 617 violent crimes per 100,000 residents, while the corresponding figure for Maine was 186 violent crimes per 100,000 residents, less than one-third of Texas's rate. The more discerning CV of the RMSPE is 0.06 for Texas (with a year of adoption discrepancy of only 3.6 percent), while for Maine, the CV is a dramatically higher at 0.196 (with an initial year discrepancy of -37.9 percent). Accordingly, since the percentage imprecision in our synthetic pretreatment match for Maine is so much greater than for Texas, we have greater confidence in our estimates that in the 10th year, Texas's RTC law had increased violent crime by 16.9 percent than we do in an estimate that Maine's law had decreased violent crime by 16.5 percent.

⁶²Ando (2015) examines the impact of constructing nuclear plants on local real per capita taxable income in Japan by generating a synthetic control for every coastal municipality that installed a nuclear plant. Although the average treatment effect measured in our article differs from the one used by Ando, we follow Ando in repeatedly estimating average placebo effects by randomly selecting different areas to serve as placebo treatments. (The sheer number of treatments that we are considering in this analysis prevents us from limiting our placebo treatment analysis to states that never adopt RTC laws, but this simply means that our placebo estimates will likely be biased *against* finding a qualitatively significant effect of RTC laws on crime, since some of our placebo treatments will be capturing the effect of the passage of RTC laws on crime rates.) Our estimated average treatment effect can then be compared to the distribution of average placebo treatment effects. Heersink and Peterson (2016) and Cavallo et al. (2013) also perform a similar randomization procedure to estimate the significance of their estimated average treatment effects, although the randomization procedure in the latter paper differs from ours by restricting the timing of placebo treatments to the exact dates when actual treatments took place.

passage dates that we use in our analysis.⁶³ For each of the 500 sets of randomly generated RTC dates, we then use the synthetic control methodology and the DAW predictors to estimate synthetic controls for each of the 33 states whose randomly generated adoption year is between 1981 and 2010. We use these data to estimate the percentage difference between each placebo treatment and its corresponding synthetic control during both the year of the treatment and each of the 10 posttreatment years (for which we have data) that follow it. Using the methodology described in notes 52 and 58, we then test whether the estimated treatment effect for each of the 10 posttreatment years is statistically significant.

To further assess the statistical significance of our results, we compare each of the 10 coefficient estimates in Table 5 with the distribution of the 500 average placebo treatment effects that use the same crime rate, posttreatment year, and sample as the given estimate. To assist in this comparison process, we report a pseudo p value that is equal to the proportion of our placebo treatment effects whose absolute value is greater than the absolute value of the given estimated treatment effect. This pseudo p value provides another intuitive measure of whether our estimated average treatment effects are qualitatively large compared to the distribution of placebo effects. Our confidence that the treatment effect that we are measuring for RTC laws is real increases if our estimated treatment effect is greater than the vast majority of our estimated average placebo treatment effects. Examining our pseudo p values in Table 5, we see that our violent crime results are always statistically significant in comparison to the distribution of placebo coefficients at the 0.05 level eight years or more past RTC adoption.

C. Synthetic Control Estimates Using LM's Explanatory Variables

In our Section III panel data analysis, we saw that RTC laws were associated with significantly higher rates of violent crime in the DAW model (Table 3), but not in the LM model (Table 4, Panel A). Under the synthetic controls approach, however, we find that the results are the same whether one uses the DAW or LM explanatory variables. This is necessarily true when one uses yearly lags in implementing the synthetic controls – see Kaul et al. (2016) – but it is also true when we use three lags of the dependent variable in our synthetic control protocol, as shown in Table 6. The detrimental effects of RTC laws on violent crime rates are statistically significant at the 0.05 level starting three years after the passage of an RTC law, and appear to increase over time. The treatment effects associated with violent crime in Table 6 range from 9.6 percent in the seventh posttreatment year to 12.8 percent in the 10th posttreatment year. Remarkably, the DAW and LM synthetic control estimates of the impact of RTC laws on violent crime are nearly identical

⁶³More specifically, we randomly choose eight states to never pass RTC laws, six states to pass RTC laws before 1981, 33 states to pass RTC laws between 1981 and 2010, and three states to pass their RTC laws between 2011 and 2014. (Washington, DC is not included in the placebo analysis since it is excluded from our main analysis.) These figures were chosen to mirror the number of states in each of these categories in our actual data set.

Table 6: The Impact of RTC Laws on the Violent Crime Rate, LM covariates, Full Sample, 1977–2014

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Average Normalized TEP	0.309 (1.318) 33	1.981 (1.646) 33	4.063* (2.192) 33	5.211* (2.572) 33	7.159** (2.887) 33	6.981** (3.319) 33	9.644*** (3.016) 33	11.160*** (3.680) 31	12.115*** (3.857) 31	12.794*** (3.200) 31
N										

NOTE: Standard errors in parentheses. Column numbers indicate post-passage year under consideration; N = number of states in sample. The synthetic controls method is run using the non-nested option, and each year's estimate and statistical significance is computed as explained in footnote 59. *, ** $p < 0.10$; ***, *** $p < 0.01$.

(compare Tables 6 and Appendix Table K1), and this is true even when we limit the sample of states in the manner described above.⁶⁴

D. The Contributions of Donor States to the Synthetic Control Estimates: Evaluating Robustness

One of the key elements of the synthetic control approach is its selection among plausible control states. For each state adopting an RTC law in year X, the approach selects among states that do not have RTC laws through at least ten years after X, including never-adopting states. Appendix Figure D10 lists all the states that are eligible under this criterion to serve as synthetic controls for one or more of the 33 adopting states, and shows how often they are selected. The horizontal length of each bar tells us how much that state contributes to our synthetic control violent crime estimates.⁶⁵ As the figure indicates, Hawaii appears most frequently—contributing to a synthetic control 18 of the 33 times it is eligible and averaging a 15.2 percent contribution—but California, a substantial contributor to multiple large states, edges it out for the largest average contribution (18.1 percent).

Hawaii's relatively large contribution as a donor state in the synthetic control estimates has some advantages but also raises concern that this small state might be unrepresentative of the states for which it is used as a control. For example, note that the largest share of Virginia's synthetic control comes from Hawaii (27.9 percent), with Rhode Island, Kansas, and Nebraska making up the lion's share of the remaining synthetic control. We had already mentioned one problem with the panel data analysis caused by the tendency of lax gun control states to serve as a source for guns that contribute to crime in the non-RTC states, and Virginia has always been a major source of that interstate flow. Since Virginia's guns are not likely to end up in Hawaii, the bias that the treatment infects the control is reduced for that particular match. Nonetheless, one may be concerned that Hawaii might be unduly skewing the estimates of the impact of RTC laws on violent crime.

To address this, as well as the analogous concern for other potentially idiosyncratic control states, we generated 18 additional TEP estimates, with each one generated by dropping a single one of the 18 states that appears as an element of our synthetic control analysis (as identified in Appendix Figure D10). The results of this exercise are presented in Appendix Figure D12, which shows that our estimated increase in violent crime resulting from the adoption of an RTC law is extremely robust: All 18 estimates remain statistically significant at the 0.01 percent level, and

⁶⁴The 10th-year effect in the synthetic control analysis using the LM variables is 12.4 percent when we eliminate the three states with more than twice the average CV of the RMSPE. Knocking out the seven states with above-average values of this CV generates a similar 12.5 percent effect.

⁶⁵In particular, it reflects the portion of each synthetic state it becomes part of, weighted by the treated state's population. For example, Texas's population is 13.6 percent of the total treated states' population. As a result, a state that made up 50 percent of synthetic Texas (but is not a donor for any other treatment state) would have a bar of size 6.8 percent.

the smallest TEP, which comes from dropping Illinois as a control state, is 12.0 percent. Note in particular that dropping Hawaii from the list of potential donor states slightly *increases* the estimate of the increase in violent crime caused by RTC laws. In fact, when we dropped Hawaii completely as a potential control and repeated the previous protocol of dropping one state at a time, the estimated increase in violent crime from RTC never fell below 12 percent (which was the value when New York was dropped as well as Hawaii). Indeed, the synthetic control finding that RTC laws increase violent crime is so robust that even if we drop California, New York, and Hawaii from the pool of potential donor states, RTC laws still increase violent crime by 8.9 percent after 10 years ($p = 0.018$).

E. Does Gun Prevalence Influence the Impact of RTC Laws?

The wide variation in the state-specific synthetic control estimates that was seen in Figures 7 and D11 suggests that there is considerable noise in some of the outlier estimates of a few individual states. For example, it is highly improbable that RTC laws led to a 16.5 percent decrease in violent crime in Maine and an 80.2 percent increase in violent crime in Montana, the two most extreme estimates seen in Figure 7. Since averaging across a substantial number of states will tend to eliminate the noise in the estimates, one should repose much greater confidence in the aggregated estimates than in any individual state estimate. Indeed, the fact that we can average across 33 separate RTC-adopting states is what generates such convincing and robust estimates of the impact of RTC laws on violent crime.

Another way to distill the signal from the noise in the state-specific estimates is to consider whether there is a plausible factor that could explain underlying differences in how RTC adoption influences violent crime. For example, RTC laws might influence crime differently depending on the level of gun prevalence in the state.

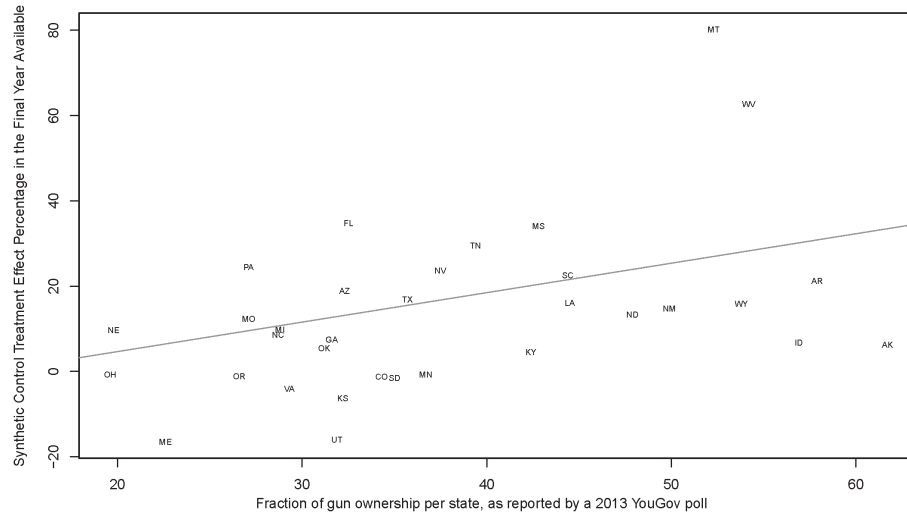
Figure 8 shows the scatter diagram for 33 RTC-adopting states, and relates the estimated impact on violent crime to a measure of gun prevalence in each RTC-adopting state. The last line of the note below the figure provides the regression equation, which shows that gun prevalence is positively related to the estimated increase in crime ($t = 2.39$).⁶⁶

F. The Murder and Property Crime Assessments with Synthetic Controls

The synthetic control estimates of the impact of RTC laws on violent crime uniformly generate statistically significant estimates, and our phantom RTC law synthetic control estimates for the five pretreatment years (described in note 58) give us confidence that the synthetic control approach is working well for our violent crime estimates, as illustrated in Appendix Table L1. Since the estimated increases in violent crime are

⁶⁶The gun prevalence data were collected by the data analytics firm YouGov in a 2013 online survey (Kalesan et al. 2016); 4,486 people were initially surveyed, although only 4,000 results are used in the final data set. YouGov used a proximity matching method to select the survey results for inclusion, matching respondents by race, age, gender, and education to the demographic breakdown of the 2010 American Community Survey.

Figure 8: The impact of gun ownership on the increase in violent crime due to RTC laws (synthetic control estimates, 1977–2014).



NOTE: Treatment effect displayed is for the 10th year after RTC adoption (but 7th post–passage year for Kansas and Nebraska). Treatment Effect = $-9.15 + 0.69 * \text{Gun Prevalence}$. $t = 2.39$; $R^2 = 0.16$. Regression weighted by population in the final TEP year.

statistically significant and consistently observed in both our panel data and synthetic control analyses, these represent our most robust finding.

Just as we saw in the panel data analysis, the synthetic controls provide evidence of increases in the murder and firearm murder categories, but it is weaker and less precise than our violent crime estimates. For example, both Appendix Tables E1 and E2 show estimated crime increases of 8.7 percent (murder) and 15.3 percent (firearm murder), but only the 8.7 figure is statistically significant at the 0.10 level. Interestingly, our phantom law test works well for murder and even suggests statistically significant increases in that crime beginning right at the time of RTC adoption (Appendix Table L3). The firearm murder estimates perform less well in this test, generating an estimated fall in crime of 6.8 percent in the year prior to RTC adoption (Appendix Table L5).

The results from implementing this phantom law approach for property crime are perhaps our less encouraging estimates. While our estimated “effect” in the year prior to adoption would ideally be close to zero in this test, for property crime it is 6.9 percent, with the latter significant at the 0.10 level. (The full results of this test for all the crime categories are shown in Appendix L.) If we accept our normalized estimate for the impact of RTC laws on property crime it would give little reason to reject a null hypothesis of no effect (Appendix Table E8). Because our synthetic control estimates for violent crime are validated by our phantom adoption test and generate uniform and highly

robust results whether dropping selected donor states or states with poor fit, or using either the DAW or LM models, we have greater confidence in and therefore highlight our violent crime estimates. Accordingly, we consign our further discussion of the synthetic control estimates of murder and property crime to Appendix E.

VI. CONCLUSION

The extensive array of panel data and synthetic control estimates of the impact of RTC laws that we present uniformly undermine the “More Guns, Less Crime” hypothesis. There is not even the slightest hint in the data from any econometrically sound regression that RTC laws reduce violent crime. Indeed, the weight of the evidence from the panel data estimates as well as the synthetic control analysis best supports the view that the adoption of RTC laws substantially raises overall violent crime in the 10 years after adoption.

In our initial panel data analysis, our preferred DAW specification predicted that RTC laws have led to statistically significant and substantial increases in violent crime. We also presented both panel data and synthetic control estimates that RTC laws substantially increase the percentage of robberies committed with a firearm, while having no restraining effect on the overall number of robberies. Moreover, to the extent the massive theft of guns from carrying guns outside the home generates crime spillovers to non-RTC states, our estimated increases in violent crime are downward biased.

We then supplemented our panel data results using our synthetic control methodology, and the finding from our panel data analysis was strongly buttressed. Whether we used the DAW or LM specifications, states that passed RTC laws experienced 13–15 percent *higher* aggregate violent crime rates than their synthetic controls after 10 years (results that were significant at either the 0.05 or 0.01 level after five years).

The synthetic control effects that we measure represent meaningful increases in violent crime rates following the adoption of RTC laws, and this conclusion remained unchanged after restricting the set of states considered based on model fit and after considering a large number of robustness checks. The consistency across different specifications and methodologies of the finding that RTC elevates violent crime enables far stronger conclusions than were possible over a decade ago when the NRC Report was limited to analyzing data only through 2000 with the single tool of panel data evaluation.

The best available evidence using different statistical approaches—panel data regression and synthetic control—with varying strengths and shortcomings and with different model specifications all suggest that the net effect of state adoption of RTC laws is a substantial increase in violent crime.

REFERENCES

- Abadie, Alberto, Alexis Diamond, & Jens Hainmueller (2010) “Synthetic Control Methods for Comparative Case Studies: Estimating the Effect of California’s Tobacco Control Program,” 105 (490) *J. of the American Statistical Association* 493.

EXHIBIT 5

Easiness of Legal Access to Concealed Firearm Permits and Homicide Rates in the United States

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Objectives. To examine the relation of “shall-issue” laws, in which permits must be issued if requisite criteria are met; “may-issue” laws, which give law enforcement officials wide discretion over whether to issue concealed firearm carry permits or not; and homicide rates.

Methods. We compared homicide rates in shall-issue and may-issue states and total, firearm, nonfirearm, handgun, and long-gun homicide rates in all 50 states during the 25-year period of 1991 to 2015. We included year and state fixed effects and numerous state-level factors in the analysis.

Results. Shall-issue laws were significantly associated with 6.5% higher total homicide rates, 8.6% higher firearm homicide rates, and 10.6% higher handgun homicide rates, but were not significantly associated with long-gun or nonfirearm homicide.

Conclusions. Shall-issue laws are associated with significantly higher rates of total, firearm-related, and handgun-related homicide. (*Am J Public Health.* 2017;107:1923–1929. doi:10.2105/AJPH.2017.304057)

 See also Donohue, p. 1864, and also Galea and Vaughan, p. 1867.

Firearm violence is a major public health problem. In 2015, there were approximately 36 000 firearm-related deaths in the United States; 13 463 were homicides, 22 018 were suicides, and 489 were unintentional injuries.¹ During the same year, 72.9% of homicides were firearm homicides¹ and, of these, approximately 90% were committed with a handgun. A central question in the debate about public policies to reduce firearm violence is whether easier access to concealed handguns increases or decreases the rate of firearm-related homicides.² Some have argued that the feared or actual presence of armed citizens may deter violent crime.³ Others have suggested that a higher prevalence of people carrying guns will increase the likelihood that an altercation results in a fatality.⁴ Thus, having a clear understanding of the impact of concealed-carry laws on firearm-related homicide would help guide policymakers who are aiming to reduce firearm violence.

As of the end of 2015, all states allowed certain persons to carry concealed handguns, but there were 3 major variations in permitting policy⁵ (Table 1). In 9 states, law

enforcement officials had wide discretion over whether to issue concealed-carry permits; these are referred to as “may-issue” states. In 32 states, there was little or no discretion; these are referred to as “shall-issue” states because permits must be issued if requisite criteria are met. In an additional 9 states, there was no permit necessary to carry a concealed handgun; these are referred to as “permitless-carry” states. The wide variation in these policies between states and over time presents the opportunity to compare homicide rates between states with varying concealed-carry permitting policies to examine the impact of concealed-carry laws on homicide.

The critical difference between may-issue and shall-issue laws is that in may-issue

states, law enforcement officials may use their judgment in making decisions about whether to approve or deny a permit application, whereas in shall-issue states, no judgment is involved—the application must be approved unless the applicant is categorically prohibited from concealed handgun possession. In may-issue states, the element of discretion allotted to law enforcement is typically a judgment regarding the “suitability” or “need” of a person to carry a concealed weapon (Table 2). Law enforcement officials have a wide degree of latitude in making these judgments. In shall-issue states, the categorical prohibitions consist of a list of specific criminal convictions.

Unfortunately, the existing literature on the impact of concealed carry laws is inconsistent. At least 10 national studies have examined the relationship between shall-issue concealed-carry laws and firearm-related or total homicide rates at the state level (Table A, available as a supplement to the online version of this article at <http://www.ajph.org>).^{3,6–14} In 2 studies, shall-issue laws were found to decrease homicide rates.^{3,6} In 2 studies, these laws were found to increase homicide rates.^{7,8} Six studies reported no clear impact of shall-issue laws on homicide rates.^{9–14} The inconsistency of these results has understandably created some confusion about what approach is most effective to address the firearm violence problem.

Most of the published literature on this topic includes data that are more than a decade old: the most recent year of data analyzed was

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TABLE 1—Concealed-Carry Permitting Laws and Age-Adjusted Firearm Homicide Rates by US State, 2015, and Status of Laws During the Period of 1991 to 2015

State	Age-Adjusted Firearm Homicide Rate, ^a 2015 (per 100 000)	Status of Concealed-Carry Permitting Law, 2015	Effective Date of Current (as of 2015) Concealed-Carry Law
Hawaii ^b	0.75	May issue	Before 1991
New Hampshire	0.96	Shall issue	Before 1991
Rhode Island	0.99	May issue	Before 1991
Maine	1.14	Shall issue	Before 1991
Massachusetts	1.26	May issue	Before 1991
Utah	1.39	Shall issue	1995
Idaho	1.29	Shall issue	Before 1991
Iowa	1.62	Shall issue	Before 1991
North Dakota	1.69	Shall issue	Before 1991
Vermont	1.76	Permitless carry	Before 1991
Minnesota	1.77	Shall issue	2003
South Dakota	1.97	Shall issue	Before 1991
New York	2.07	May issue	Before 1991
Wyoming	2.16	Permitless carry	2011 ^c
Montana	2.17	Shall issue	Before 1991
Washington	2.32	Shall issue	Before 1991
Oregon	2.35	Shall issue	Before 1991
Connecticut	2.43	May issue	Before 1991
Colorado	2.46	Shall issue	2003
Nebraska	2.67	Shall issue	2007
West Virginia	2.89	Shall issue	Before 1991
Wisconsin	3.18	Shall issue	2011
New Jersey	3.22	May issue	Before 1991
Virginia	3.29	Shall issue	1995
Kansas	3.35	Shall issue	2007
California	3.52	May issue	Before 1991
Arizona	3.56	Permitless carry	2010 ^c
Kentucky	3.96	Shall issue	1996
Texas	4.04	Shall issue	1995
Pennsylvania	4.34	Shall issue	Before 1991
Ohio	4.38	Shall issue	2004
Nevada	4.49	Shall issue	1995
North Carolina	4.54	Shall issue	1995
Indiana	4.61	Shall issue	Before 1991
Florida	4.66	Shall issue	Before 1991
Michigan	4.74	Shall issue	2001
New Mexico	4.79	Shall issue	2001
Alaska	5.22	Permitless carry	2003 ^c

Continued

2010, and only 3 of the 10 studies examined data past the year 1998 (Table A, available as a supplement to the online version of this article at <http://www.ajph.org>). Since 1998, 11 additional states have enacted shall-issue laws.⁵ This provides more variation over time and a longer follow-up period to examine this research question. Moreover, Ayres and Donohue¹⁵ and Hepburn et al.¹¹ have suggested that the relationship between concealed-carry laws and homicide rates may have been different during the period before and after the early 1990s. In addition, studies that included homicide rates from before 1994 were examining a trend that was increasing, whereas studies examining homicide rates after 1994 were capturing declining trends. For these reasons, a reexamination of this research question with more recent data is needed.

One limitation of the existing literature is that no previously published research has examined the specific impact of concealed-carry laws on handgun versus long-gun homicide rates. This is important because if such laws increase homicide by making it easier for people at high risk for violence to carry handguns, this effect should only be observed in relation to handgun-related homicides, not homicides committed with long guns. On the other hand, if permissive concealed-carry laws deter crime by generating fear among potential perpetrators of encountering an armed individual, then all crime including handgun, long-gun, and nonfirearm homicide should decrease.

Another limitation of previous studies is that nearly all of them used linear models. However, homicide rates represent count data, and the distribution of homicide rates across states is highly skewed¹⁶ (Figure A, available as a supplement to the online version of this article at <http://www.ajph.org>). Plassmann and Tideman argued that a count model (such as a Poisson or negative binomial model) is the most reliable for analyzing crimes, such as homicides, with low occurrence rates.¹⁶ Beyond the Plassmann and Tideman study, only 1 other study¹¹ used a count model.

We examined the relationship between shall-issue concealed-carry laws and total, firearm-related, and non-firearm-related homicide rates, as well as handgun versus long-gun homicide rates across all 50 states

TABLE 1—Continued

State	Age-Adjusted Firearm Homicide Rate, ^a 2015 (per 100 000)	Status of Concealed-Carry Permitting Law, 2015	Effective Date of Current (as of 2015) Concealed-Carry Law
Arkansas	5.34	Shall issue	1995
Illinois	5.45	Shall issue	2013
Tennessee	5.51	Shall issue	1994
Georgia	5.73	Shall issue	Before 1991
Oklahoma	5.87	Shall issue	1995
Delaware	6.12	May issue	Before 1991
South Carolina	7.55	Shall issue	1996
Maryland	7.69	May issue	Before 1991
Missouri	7.92	Shall issue	2003
Alabama	8.43	Shall issue	2013
Mississippi	9.11	Shall issue	1991
Louisiana	9.96	Shall issue	1996

Note. "May-issue" states are those in which law enforcement officials had wide discretion over whether to issue concealed-carry permits. "Shall-issue" states are those in which there was little or no discretion; permits must be issued if requisite criteria are met. "Permitless-carry" states are those in which there was no permit necessary to carry a concealed handgun.

^aFrom Centers for Disease Control and Prevention (CDC).¹

^bData for Hawaii are unavailable for the years 2010 to 2015 because the CDC's Web-Based Injury Statistics Query and Reporting Systems does not report homicide counts fewer than 10. The data here are from 2009.

^cChanged from "may issue" to "shall issue" in 1994.

during the 25-year time period of 1991 to 2015 with both count and linear regression models. We examined the specificity of the relationship between concealed-carry laws and homicide rates by separately modeling firearm versus nonfirearm homicide rates and then within firearm-related homicides by modeling handgun versus long-gun homicide rates. We analyzed the relationship between shall-issue concealed-carry laws and homicide rates by using both a count and a linear regression model, thus examining the robustness of results to the type of model used.

METHODS

We used a quasi-experimental panel design, taking advantage of changes in state concealed-carry permitting laws over time, to explore the relationship between these laws and total, firearm-related, and non-firearm-related homicide rates in the 50 states over a 25-year period, 1991 to 2015. We

modeled homicide rates in 2 ways: (1) using a negative binomial regression with homicide rates as the outcome variable and (2) using linear regression with log-transformed homicide rates as the outcome variable. In both cases, we included year and state fixed effects and controlled for a range of time-varying, state-level factors.

Variables and Data Sources

Outcome variables. The main outcome variable was the age-adjusted firearm homicide rate in each year analyzed. For example, Missouri's shall-issue law went into effect in 2003; thus, we analyzed homicide rates associated with Missouri's shall-issue law for the years 2004 to 2015. We obtained homicide rates from the Centers for Disease Control and Prevention's (CDC's) Web-Based Injury Statistics Query and Reporting Systems (WISQARS) database.¹ This is the ideal source for homicide data because there is complete annual reporting from all 50 states and because the data are extracted from the

Vital Statistics death registry maintained by the National Center for Health Statistics, which is based on standardized death certificates. The completeness of reporting is approximately 99%.¹⁷ The CDC age-adjusted the rates to the 2000 standard population.

The second outcome variable was the handgun or long-gun homicide rate, obtained from the Federal Bureau of Investigation's Uniform Crime Reports, Supplemental Homicide Reports (SHR).¹⁸ Although WISQARS does provide mortality data from *International Classification of Diseases, Ninth Revision* and *Tenth Revision*, codes that can list handgun and long gun as the cause of death, unfortunately, most death certificates involving a firearm homicide do not specify the type of weapon used. Therefore, most firearm homicide deaths in WISQARS are classified as "other and unspecified" firearm, and it is not possible to use these data to disaggregate handgun and long-gun homicides.¹⁹ By contrast, the SHR is missing data on the type of weapon used in firearm homicides in just 13.4% of cases. Thus, the SHR is the best, if not only, source for state-specific, firearm type-specific homicide data.

The SHR disaggregates firearm homicides into handgun, rifle, shotgun, and other (and unknown). We used the handgun deaths to generate handgun homicide rates and the sum of rifle, shotgun, and other gun deaths to generate long-gun homicide rates for each state and year. Although SHR data may include listing of multiple weapons in an incident, only 1 weapon may be associated with a homicide death.²⁰ Because of missing data on weapon type, we excluded 13.4% of firearm homicide cases in estimating handgun homicide rates. Nevertheless, there was little discrepancy between the firearm homicide totals from WISQARS and the SHR, which were correlated at $r = 0.98$.

Because not all local law enforcement agencies complete the supplemental reports, the SHR data set excludes approximately 10% of all homicides.²¹ This problem was addressed by applying weights that adjusted each state- and year-specific estimate up to the overall number of homicides reported in the Uniform Crime Report for that state and year. Fox kindly provided us with updated SHR files that added previously

TABLE 2—Elements of Discretion in Law Enforcement Decisions to Approve or Deny Concealed Handgun Carry Permits: “May-Issue” US States, 2015

State	Elements of Discretion	Citation
California	Applicant must be of “good moral character” and must have “good cause” for issuance of the license.	California Penal Code § 26150, § 26155
Connecticut	Applicant must intend only to make “legal use” of the handgun and must be a “suitable person to receive such permit.”	Connecticut General Statutes § 29-28
Delaware	Applicant must be “of good moral character,” must desire the handgun for “personal protection” or “protection of the person’s property,” and must submit signed, written statements of 5 “respectable citizens” of the county who testify that the applicant is a person “of sobriety and good moral character” and “bears a good reputation for peace and good order in the community” and that a handgun is “necessary for the protection of the applicant or the applicant’s property.” The Superior Court has discretion to approve or deny the application.	Delaware Code § 1441
Hawaii	Must be “an exceptional case,” the applicant must show “reason to fear injury to the applicant’s person or property,” the applicant must be “a suitable person” to be licensed, and the chief of police must determine that the person “is qualified to use the firearm in a safe manner.”	Hawaii Revised Statutes § 134-9
Maryland	Applicant must have a “good and substantial reason to wear, carry, or transport a handgun, such as a finding that the permit is necessary as a reasonable precaution against apprehended danger,” and the applicant must not have “exhibited a propensity for violence or instability that may reasonably render the person’s possession of a handgun a danger to the person or to another.”	Maryland Public Safety Code § 5-306
Massachusetts	Applicant must be a “suitable” person and must not be judged to potentially create a risk to public safety.	Massachusetts General Laws 140 § 131
New Jersey	Applicant must demonstrate a “justifiable need to carry a handgun” and must submit endorsements by 3 individuals who have known the applicant for at least 3 years that the applicant is “a person of good moral character and behavior.”	New Jersey Statutes § 2C:58-4
New York	Applicant must be “of good moral character,” must be “of good character, competency, and integrity,” and there must be no “good cause” for denial of the license.	New York Penal Law § 400.00
Rhode Island	Applicant must have “good reason to fear an injury to his or her person or property” or have “any other proper reason” for carrying a handgun and must be a “suitable person to be so licensed.”	General Laws of Rhode Island § 11-47-11

Note. “May-issue” states are those in which law enforcement officials had wide discretion over whether to issue concealed-carry permits.

missing data for Florida and included data through 2015.²¹

Main predictor variable. Using *Thomson Reuters Westlaw* to access historical state statutes and session laws, we developed a database indicating the presence or absence of 100 provisions of firearm laws in each state over the 25-year period.⁵ We coded laws by the year they went into effect, regardless of the month of the effective date. However, in the analytic models, we lagged the state laws by 1 year, which ensured that all laws were in effect during the year in which their impact was being assessed. Following Lott and Mustard,²² we assessed the impact of laws starting in the first full year they were in effect.

We examined the potential impact of shall-issue laws, comparing them to may-issue laws. In other words, using the may-issue states as the reference group, we

estimated the impact of shall-issue laws on homicide rates. Because only 4 states had permitless-carry laws in place during the study period, there were not enough observations to allow any meaningful analyses of these laws. Therefore, we deleted state-year observations in which a permitless-carry law was in effect.

Control variables. We controlled for 12 state-level factors that (1) were found in the previous literature^{3,6–14} to be significantly related to homicide rates and (2) were significantly related to the presence of shall-issue laws in our data set (i.e., the regression coefficient for the variable was significant at a level of $P = .05$ in a logistic regression with shall-issue law as the dependent variable): household firearm ownership (using the standard proxy, which is the percentage of all suicides committed with a firearm), proportion of Blacks, proportion of young adults

(aged 18 to 29 years), proportion of men among young adults, proportion of the population living in urban areas, total population, population density, per capita alcohol consumption, the nonhomicide violent crime rate (aggravated assault, robbery, and forcible rape), the poverty rate, unemployment rate, median household income, per capita disposable income, incarceration rate, and per capita number of law enforcement officers. Variable definitions and data sources are provided in Table B, available as a supplement to the online version of this article at <http://www.ajph.org>. We also controlled for the following state firearm laws that could serve as alternative explanations for changes in homicide during the study period: (1) universal background checks required for all handgun purchases, (2) waiting periods required for all handgun purchases, and (3)

permits required to purchase or possess firearms.

Analysis

Count models. Because homicide rates are not normally distributed but skewed and overdispersed, we modeled this outcome by using a negative binomial distribution. To control for clustering in our data by year (25 levels) and by state (50 levels), we entered year and state as fixed effects in the regression models. We used robust standard errors that account for the clustering of observations, serial autocorrelation, and heteroskedasticity.²³

Our final model was as follows:

$$(1) \Pr(H_{st} = h_{st}) = \frac{\Gamma(y_{st} + \alpha^{-1})}{\Gamma(y_{st} + 1)\Gamma(\alpha^{-1})} [1 / (1 + \alpha \mu_{st})]^{1\alpha} \left[\mu_{st} / (\alpha^{-1} + \mu_{st}) \right]^{y_{st}},$$

where $\Pr(H_{st} = h_{st})$ is the probability that state s in year t has a homicide rate equal to h_{st} , $E(H_{st}) = \mu_{st}$, and $\text{Var}(H_{st}) = \mu_{st} + \mu_{st}^2$.

The mean homicide rate was then modeled as follows:

$$(2) \ln(\mu_{st}) = \alpha + \beta_1 CC_{st} + \beta_2 C_{st} + S + T + e,$$

where CC_{st} is a dummy variable for the presence of a shall-issue law, C is a vector of control variables, S represents state fixed effects, and T represents year fixed effects.

The negative binomial regression coefficients are reported as incidence rate ratios (IRRs). The IRR indicates the percentage difference in homicide rate for states with a shall-issue concealed-carry law compared with states with a may-issue law.

Linear models. To check the robustness of our findings, we repeated the analyses with a linear regression model, with the log-transformed homicide rate as the outcome variable, again by using robust standard errors.²³ As with the negative binomial models, we included year and state fixed effects, and we included the same state-level control variables.

We conducted analyses with Stata version 14.1 (StataCorp LP, College Station, TX).

We evaluated the significance of regression coefficients by using a Wald test at $\alpha = 0.05$.

We checked the robustness of our results by conducting several sensitivity analyses, including

1. Restricting the analysis to the 23 states in which shall-issue laws were adopted during the study period,
2. Using raw count data instead of homicide rates,
3. Restricting the analysis to states with population greater than 1 000 000,
4. Restricting the analysis to the period 1991 to 2002,
5. Restricting the analysis to the period 2003 to 2015, and
6. Using SHR instead of WISQARS homicide data (thus avoiding the problem of missing data for some smaller states after 1998).

RESULTS

During the study period, 23 states adopted shall-issue laws (Table 1). By 2015, 37 states had such laws. In the same year, the average firearm homicide rate in the states with shall-issue laws was 4.11 per 100 000, compared with 3.41 per 100 000 in the may-issue states. The number of states that had permitless-carry laws in effect at all during the study period was small ($n = 4$), as was the number of observations ($n = 46$), limiting our ability to analyze the impact of these laws. Because CDC does not report homicide counts of fewer than 10 in years after 1998, we were missing outcome data for several years for 6 states (Hawaii, New Hampshire, North Dakota, South Dakota, Vermont, and Wyoming); a sensitivity analysis with SHR data revealed that these omissions do not affect our findings.

In negative binomial regression models, shall-issue concealed-carry permitting laws were significantly associated with 6.5% higher total homicide rates compared with may-issue states (IRR = 1.065; 95% confidence interval [CI] = 1.032, 1.099; Table 3). The association was specific to firearm homicide rates, which were 8.6% higher in shall-issue states (IRR = 1.086; 95% CI = 1.047, 1.126). There was no significant

association between shall-issue laws and nonfirearm homicide rates (IRR = 1.014; 95% CI = 0.963, 1.068). Further disaggregation within firearm homicides showed that the association between shall-issue laws and firearm homicide rates was specific to handgun homicide. Shall-issue states had handgun homicide rates that were 10.6% higher (IRR = 1.106; 95% CI = 1.039, 1.177), but there was no significant association with long-gun homicide rates (IRR = 0.999; 95% CI = 0.915, 1.090).

The results of the linear regression analyses were similar. Here, shall-issue laws were significantly associated with 6.6% higher total homicide rates compared with may-issue states (95% CI = 3.0%, 10.4%; data not shown). The association was specific to firearm homicide rates, which were 11.7% higher in “shall issue” states (95% CI = 6.4%, 17.2%); there was no significant association between these laws and nonfirearm homicide rates. Further disaggregation within firearm homicides showed that the association between shall-issue laws and firearm homicide rates was specific to handgun homicide. Shall-issue states had handgun homicide rates that were 19.8% higher (95% CI = 10.3%, 30.1%), but rates of long-gun homicide were not significantly different in states with shall-issue compared with may-issue laws.

The significant association between shall-issue laws and higher total, firearm, and handgun-related homicide rates remained when we restricted the analysis to the 23 states in which these laws were adopted during the study period (Table 3). This pattern of results was robust to a series of additional sensitivity checks, including using raw count data, restricting the analysis to states with a population of more than 1 000 000, restricting the analysis to the period 1991 to 2002, restricting the analysis to the period 2003 to 2015, and using SHR instead of WISQARS homicide data.

DISCUSSION

To the best of our knowledge, this is the first study to examine the relationship between concealed-carry permitting laws and handgun-specific homicide rates. We found that, when we used both count and linear

models and after we controlled for a range of time-varying state factors and for unobserved time-invariant state factors by using a fixed-effects model, shall-issue concealed-carry permitting laws were significantly associated with 6.5% higher total homicide rates, 8.6% higher firearm-related homicide rates, and 10.6% higher handgun-specific homicide rates compared with may-issue states.

A major reason for inconsistent results in the existing literature on the effects of concealed-carry laws may be that the relationship between concealed-carry laws and homicide rates was different during the period before and after the early 1990s.^{11,15} It is possible that despite the enactment of early shall-issue laws in the 1970s and 1980s, the demand for handgun permits in those states was modest. There has been a striking increase in the demand for pistols, especially those designed for concealed carry, during the past decade.²⁴ Recently, Steidley found that the adoption of shall-issue laws during the period 1999 to 2013 was associated with a persistent, long-term increase in handgun sales in all 7 states studied.²⁵ Our analysis provides further support for the hypothesis that the relationship between shall-issue laws and higher homicide rates increased over time, as the regression coefficients for these laws was higher for the second half of the study period

(2003–2015) compared with the first half (1991–2002).

Our finding that the association between shall-issue laws and homicide rates is specific to handgun homicides adds plausibility to the observed relationship. If the relationship between shall-issue laws and homicide rates were spurious, one might expect to see the relationship hold for long-gun as well as handgun homicide rates. Moreover, this finding is inconsistent with the hypothesis that permissive concealed-carry laws deter crime by increasing the presence of armed individuals. Were that the case, one would expect to see lower handgun, nonhandgun, and nonfirearm homicide rates in shall-issue compared with may-issue states. The lack of an association between shall-issue laws and long-gun homicide rates is also inconsistent with the hypothesis that the presence of more concealed weapons escalates the level of violence in encounters that may involve a long gun.

Strengths and Limitations

This study has several novel strengths, including the use of both count and linear models, the use of recent data (through 2015), and the disaggregation of homicide rates. Nevertheless, caution should be exercised in assessing causality from an ecological study

such as this one. In particular, these results should be interpreted with caution because of the possibility that they reflect a reverse association. That is, it is possible that the adoption of shall-issue concealed carry laws is associated with higher baseline homicide rates so that we are picking up not a causal effect of these laws on homicide but a systematic difference in baseline homicide rates between states that do or do not have these laws. However, our findings hold even when the analysis is restricted to states that started with may-issue laws at the beginning of the study period and adopted shall-issue laws during the study period.

An additional limitation of this study is that we could not consider the enforcement of concealed-carry laws.²⁶ Enforcement of these laws may vary not only among states, but also among counties in the same state.¹¹ In addition, we did not have information on the number of concealed-carry permits issued in each state or the number of homicides committed by concealed-carry permittees.

It is also important to note that we examined only fatal firearm injuries. Further research should investigate potential effects of concealed-carry laws on nonfatal firearm injuries.

Finally, we were unable to analyze the impact of permitless-carry laws because of the small number of observations. Only 4 states

TABLE 3—Sensitivity Analyses of Relationship Between “Shall-Issue” Concealed-Carry Permitting Laws and Homicide Rates: United States, 1991–2015

Type of Analysis	Homicide Rate, IRR (95% CI)		
	Total	Firearm	Handgun
Main analysis	1.065 (1.032, 1.099)	1.086 (1.047, 1.126)	1.106 (1.039, 1.177)
Analysis restricted to states that adopted shall-issue concealed-carry laws during study period	1.063 (1.028, 1.099)	1.068 (1.030, 1.108)	1.074 (1.002, 1.150)
Analysis using raw count of homicides with population as the exposure variable	1.051 (1.020, 1.083)	1.079 (1.039, 1.120)	1.139 (1.067, 1.217)
Analysis restricted to states with population > 1 million	1.055 (1.023, 1.087)	1.067 (1.030, 1.105)	1.095 (1.029, 1.166)
Analysis restricted to years before 2003 (1991–2002)	1.058 (1.014, 1.104)	1.067 (1.019, 1.116)	1.107 (1.037, 1.180)
Analysis restricted to years after 2002 (2003–2015)	1.064 (1.009, 1.122)	1.100 (1.028, 1.176)	1.274 (1.092, 1.488)
Analysis using Supplemental Homicide Report data instead of Vital Statistics data	1.044 (1.006, 1.083)	1.094 (1.047, 1.143)	1.106 (1.039, 1.177)

Note. “Shall-issue” states are those in which there was little or no discretion; permits must be issued if requisite criteria are met. CI = confidence interval; IRR = incidence rate ratio. All models include year and state fixed effects and control for the following time-varying, state-level factors: household gun-ownership levels, proportion of young men, proportion of young adults, proportion of Blacks, proportion living in an urban area, total population, population density, median household income, poverty rate, unemployment rate, per capita disposable income, per capita alcohol consumption, violent crime rate, incarceration rate, per capita law enforcement officers, universal background check laws for all handguns, waiting periods for all handguns, and permits required for all firearms.

had permitless-carry laws in place during the study period. However, in the past 2 years, an additional 5 states have enacted such laws. Elucidating the impact of permitless-carry laws will require follow-up for the 9 states that now have such laws in effect.

Conclusions

Despite these limitations, this study suggests that there is a robust association between shall-issue laws and higher rates of firearm homicides. The trend toward increasingly permissive concealed-carry laws is inconsistent with public opinion, which tends to oppose the carrying of guns in public.²⁷ Our findings suggest that these laws may also be inconsistent with the promotion of public safety. *AJPH*

CONTRIBUTORS

M. Siegel conceptualized the study, led the data analysis and writing, and was the principal author of this article. Z. Xuan and C. S. Ross assisted with the study design and analytical plan. All authors contributed toward the interpretation of data analyses, critical review of the article, and revision of the article.

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Note. The views expressed here do not necessarily reflect those of the Robert Wood Johnson Foundation.

HUMAN PARTICIPANT PROTECTION

This study made use of secondary data only and did not require institutional review board approval.

REFERENCES

- Centers for Disease Control and Prevention. Web-Based Injury Statistics Query and Reporting Systems: fatal injury reports. Available at: http://www.cdc.gov/injury/wisqars/fatal_injury_reports.html. Accessed March 15, 2017.
- Donohue JJ. Guns, crime, and the impact of state right-to-carry laws. *Fordham Law Rev.* 2005;73:623–652.
- Lott JR. *More Guns, Less Crime: Understanding Crime and Gun Control Laws*. 3rd ed. Chicago, IL: The University of Chicago Press; 2010.
- Miller M, Azrael D, Hemenway D. Firearms and violent death in the United States. In: Webster DW, Vernick JS, eds. *Reducing Gun Violence in America: Informing Policy With Evidence and Analysis*. Baltimore, MD: The Johns Hopkins University Press; 2013.
- Siegel M, Pahn M, Xuan Z, et al. Firearm-related laws in all 50 states, 1991–2016. *Am J Public Health.* 2017; epub ahead of print May 18, 2017.
- Lott JR Jr, Whitley JE. Safe-storage gun laws: accidental deaths, suicides, and crime. *J Law Econ.* 2001;44: 659–689.
- Zimmerman PR. The deterrence of crime through private security efforts: theory and evidence. *Int Rev Law Econ.* 2014;37:66–75.
- Ludwig J. Concealed-gun-carrying laws and violent crime: evidence from state panel data. *Int Rev Law Econ.* 1998;18:239–254.
- Aneja A, Donohue JJ, Zhang A. The impact of right-to-carry laws and the NRC report: lessons for the empirical evaluation of law and policy. *Am Law Econ Rev.* 2011; 13(2):565–632.
- Rosengart M, Cummings P, Nathens A, Heagerty P, Maier R, Rivara F. An evaluation of state firearm regulations and homicide and suicide death rates. *Inj Prev.* 2005;11(2):77–83.
- Hepburn L, Miller M, Azrael D, Hemenway D. The effect of nondiscretionary concealed weapon carrying laws on homicide. *J Trauma.* 2004;56(3):676–681.
- Sommers PM. Deterrence and gun control: an empirical analysis. *Atl Econ J.* 1980;8:89–94.
- DeZee MR. Gun control legislation: impact and ideology. *Law Policy Q.* 1983;5(3):367–379.
- Murray DR. Handguns, gun control laws and firearm violence. *Soc Probl.* 1975;23:81–93.
- Ayres I, Donohue JJ III. Shooting down the “more guns, less crime” hypothesis. *Stanford Law Rev.* 2003;55: 1193–1300.
- Plassmann F, Tideman TN. Does the right to carry concealed handguns deter countable crimes? Only a count analysis can say. *J Law Econ.* 2001;44:771–798.
- Regoece W, Banks D. *The Nation's Two Measures of Homicide*. Washington, DC: US Department of Justice, Office of Justice Programs, Bureau of Justice Studies; 2014.
- National Archive of Criminal Justice Data. *Uniform Crime Reporting Program Data Series. Supplemental Homicide Reports, 1981–2015*. Ann Arbor, MI: Inter-university Consortium for Political and Social Research; 2016.
- Rokaw WM, Mercy JA, Smith JC. Comparing death certificate data with FBI crime reporting statistics on US homicides. *Public Health Rep.* 1990;105(5):447–455.
- Supplementary homicide report (OMB form no. 1110–0002), offense 1a. Murder and nonnegligent manslaughter. Washington, DC: Federal Bureau of Investigation; 2017. Available at: <https://ucr.fbi.gov/nibrs/addendum-for-submitting-cargo-theft-data/shr>. Accessed June 17, 2017.
- Fox J. Multiply-imputed supplementary homicide reports file, 1976–2015. Boston, MA: Northeastern University; 2017.
- Lott JR, Mustard DB. Crime, deterrence, and right-to-carry concealed handguns. *J Legal Stud.* 1997;26:1–68.
- White H. A heteroskedasticity-consistent covariance matrix estimator and a direct test for heteroskedasticity. *Econometrica.* 1980;48(4):817–838.
- Smith VM, Siegel M, Xuan Z, et al. Broadening the perspective on gun violence: an examination of the firearms industry, 1990–2015. *Am J Prev Med.* 2017; Epub ahead of print.
- Steidley T. *Movements, Malefactions, and Munitions: Determinants and Effects of Concealed Carry Laws in the United States* [dissertation]. Columbus, OH: The Ohio State University; 2016.
- Lott JR. Not all right-to-carry laws are the same, yet much of the literature keeps ignoring the differences. Crime Prevention Research Center. 2014. Available at: <https://ssrn.com/abstract=2524729>. Accessed April 15, 2017.
- Wolfson JA, Teret SP, Azrael D, Miller M. US public opinion on carrying firearms in public places. *Am J Public Health.* 2017;107(6):929–937.

EXHIBIT 6



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Using the synthetic control method to determine the effects of concealed carry laws on state-level murder rates

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ABSTRACT

The purpose of the present study is to determine the relationship between concealed carry (CCW) laws and state-level murder rates. Specifically, this study will examine the impact of a change in CCW status from “prohibited” to “shall issue” on murder rates. Using a synthetic control method, results of the present study suggest that only in New Mexico did the move from “prohibited” CCW status to “shall issue” CCW status result in an increase in murder rates and gun related murder rates. For the remaining states, the change in CCW status had no effect on murder rates. As a robustness check on the results found using the synthetic control method, a fixed effects model was also estimated. These results indicate that states that changed from “prohibited” to “shall issue” experienced a 12.3% increase in gun-related murder rates and a 4.9% increase in overall murder rates. It is important to note that none of the results in the present study indicate that a move from “prohibited” to “shall issue” CCW status may result in a decline in murder rates.

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1. Introduction

There is a common belief among gun rights proponents that firearms should be readily available to the general public in order to promote deterrence. According to this argument, criminals would be less likely to commit crimes if they believed that their victims or innocent bystanders may be armed. This deterrent effect has been discussed at great length in many prior studies (Kovandzic and Marvell, 2003; Kovandzic et al., 2005; Lott and Mustard, 1997). Gun control proponents, however, believe that, by increasing the supply and availability of firearms, the probability that a crime will be committed also increases. Most gun control proponents fear that minor altercations may become violent and deadly if more people are armed.

One of the more prevalent and important types of gun control measures that may affect the overall availability of firearms is the concealed carry law. Concealed carry (CCW) refers to the carrying of firearms in a concealed fashion. Laws regulating the concealed carry of firearms differ substantially between states and have changed quite significantly over the past thirty years. There are no federal laws regulating the concealed carry of firearms.

There are four broad types of state-level CCW laws (Barati, 2016; Donohue, 2003; Gius, 2014). The first is “unrestricted”; individuals

in states with unrestricted access do not need a permit to carry a concealed handgun. For years, the only state that had no CCW restrictions was Vermont. The next type of CCW law is “shall issue”. In a “shall issue” state, a permit is required to carry a concealed weapon, but state and local authorities must issue a permit to any qualified applicant who requests one. “Shall issue” and unrestricted are considered to be permissive CCW laws.

The third type of CCW law is “may issue.” In a “may issue” state, local and state authorities can deny requests for concealed carry permits, even requests from qualified applicants. “May issue” laws restrict the ability of citizens to carry concealed weapons. Finally, the last category is “prohibited”; in years past, some states prohibited the concealed carry of firearms. As of 2018, there are no states that prohibit concealed carry.

Presently, the following nine states and the District of Columbia require a permit to carry a concealed handgun and are “may issue”: California, Connecticut (per statute; in practice “shall issue”), Delaware, Hawaii, Maryland, Massachusetts, New Jersey, New York, and Rhode Island (Barati, 2016; Donohue, 2003; Gius, 2014). The following six states do not require a permit to carry a concealed handgun: Alaska, Arizona, Kansas, Maine, Vermont, and Wyoming (Barati, 2016; Donohue, 2003; Gius, 2014). All other states require a permit and are “shall issue” (Barati, 2016; Donohue, 2003; Gius, 2014).

It is important to note that these four categories of CCW laws are rather broad, and not all states within a given category are equally

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Table 1

Summary of Papers that Examined Effects of Concealed Carry Laws on Violent Crime Rates.

	Permissive CCW Laws Reduce Crime	Permissive CCW Laws Increase Crime	Insignificant or Mixed Results
Ayers and Donohue, 2003			X
Barati, 2016			X
Bartley and Cohen, 1998	X		
Benson and Mast, 2001	X (Model Specific)		
Black and Nagin, 1998			X
Blau et al., 2016			X
Bronars and Lott, 1998	X		
Crifasi et al., 2016			X
Dezhbakhsh and Rubin, 1998			X
Donohue, 2003			X
Duwe et al., 2002			X
Ginwalla et al., 2013		X	
Gius, 2014	X		
Helland and Tabarrok, 2004	X		
Kleck and Patterson, 1993			X
Kovandzic and Marvell, 2003			X (Only Florida)
Kovandzic et al., 2005			X
Lott, 1998	X		
Lott and Mustard, 1997	X		
Ludwig, 1998		X	
Moody, 2001	X		
Moody et al., 2014	X		
Olson and Maltz, 2001	X		
Phillips et al., 2015			X
Plassman and Tideman, 2001	X		
Rubin and Dezhbakhsh, 2003			X
Shi and Lee, 2017			X
Smith and Petrocelli, 2018			X

restrictive. These broad categories are not definitive because the way in which a state not only interprets CCW statutes but also enforces them has an impact on the actual restrictiveness of the law. In addition, some cities and counties have more restrictive concealed carry laws than their home states. Finally, it is important to note that permits to carry concealed firearms are required in both “shall issue” and “may issue” states. The only difference between these two types of CCW statutes is that, in “shall issue” states, the issuing authority must issue a CCW permit to any qualified applicant. In a “may issue” state, the issuing authority may deny a permit to anyone, even qualified applicants. Hence, the vast majority of states in the U.S. require a permit to carry a concealed weapon.

Regarding CCW research, Table 1 summarizes the findings of most of the research in this area over the past 20 years. As can be seen from this table, most studies found that permissive concealed carry laws (“shall issue” or unrestricted) either reduce violent crime or they have no significant effects on crime. Only two studies (Ginwalla et al., 2013; Ludwig, 1998) found that permissive concealed carry laws increase homicide rates. Some studies, such as Ayers and Donohue (2003), Donohue (2003), and Kovandzic and Marvell (2003), found that states with more permissive concealed carry laws had higher property crime rates. However, Devaraj and Patel (2018) found that property crime rates fell when Chicago went from “prohibited” CCW status to “shall issue”. Hence, most prior research has found that permissive CCW laws either reduce violent crime, or that they have no statistically significant effects on violent crime.

In one of the earliest studies on CCW laws, Lott and Mustard (1997) found that states with “shall issue” concealed carry laws had lower crime rates than states with more restrictive carry laws. They found that “shall issue” laws resulted in a 7.65% drop in murders and a 5% drop in rapes. Their research suggests that individuals would be less likely to commit crimes if they knew that others may be carrying concealed weapons. Much of the research that followed Lott

and Mustard (1997) used the same type of model (log-linear) and the same type of data (county-level). In addition, most of these studies used panel data estimation techniques, weighted observations, and clustered standard error corrections.

One of the most recent studies on the relationship between CCW laws and crime is Barati (2016). In this study, the author used a difference-in-differences model in order to determine the effects of a change in state-level CCW status on various crime rates. Specifically, Barati (2016) attempted to determine if the effects of a “shall issue” CCW law differed depending on what type of law was in effect prior to the passage of the “shall issue” law. Using data for the period 1991–2008, the author found that switching from a “may issue” law to a “shall issue” law had no statistically-significant effects on any of the crime rates examined. However, when a state switched from prohibited to “shall issue”, then robbery, burglary, and larceny rates all fell. The change from prohibited to “shall issue” had no statistically-significant effects on motor vehicle thefts, murders, or aggravated assaults. A similar approach regarding the effects of a change in CCW status from “prohibited” to “shall issue” is employed in the present study.

The purpose of the present study is to determine the relationship between concealed carry laws and state-level murder rates. Specifically, this study will examine the impact of the change in CCW status from “prohibited” to “shall issue” on murder rates. The reason why this change is important is because, over the past 30 years, many states have loosened their CCW laws and have changed from “prohibited” to “shall issue”. In addition, as noted earlier, both “may issue” and “shall issue” states require permits for concealed carry. Therefore, a change from “may issue” to “shall issue” is not as significant nor as noteworthy as a change from “prohibited” to “shall issue”. Also, very few states, even today, allow unrestricted concealed carry. Hence, examining the impact of a shift to “unrestricted” carry on crime would be somewhat problematic. Therefore, the present study will attempt to determine the relationship between changes in CCW laws and state-level murder rates.

Another important distinction of the present study is that a synthetic control method will be used to examine the impact of this change in CCW laws. In most prior studies on this topic, a fixed effects model with panel data was used to estimate the relationship between gun control laws and crime rates. Although well-established in the area of policy analysis, fixed effects may not be the most appropriate statistical method to use in the analysis of the effects of gun control measures on crime. The results are highly dependent upon the time period being examined, and misspecification of the model may make it difficult to establish causal relationships between gun control laws and crime (Donohue et al., 2017). A superior and more appropriate statistical method for this type of analysis may be the synthetic control method. Only one other study used the synthetic control method to examine the impact of a change in CCW laws on crime rates (Donohue et al., 2017).

Results of the present study suggest that only in New Mexico did the move from “prohibited” to “shall issue” result in an increase in murder rates and gun-related murder rates. For the remaining states, the change in CCW status had no effects on murder rates or the results were inconclusive. As a robustness check on the results found using the synthetic control method, a fixed effects model was also estimated. These results suggest that states that shifted from “prohibited” to “shall issue” experienced a 12.3% increase in gun-related murder rates and a 4.9% increase in overall murder rates. It is important to note that neither the synthetic control method nor the traditional fixed effects model found that a move to more permissive CCW laws resulted in fewer murders.

2. Empirical technique and data

In order to determine if CCW laws are related to murder rates, a synthetic control method (SCM) is used in the present study. The synthetic control method (SCM) examines how a treatment (law) can affect a particular outcome (murder rates). In an SCM, there is one individual or entity (state) that receives the treatment (treated group) and several entities that do not receive the treatment (control group). The SCM then synthesizes a control from a weighted sum of potential control entities. The outcome variable for the treated group is then compared to the outcome variable for the synthesized control group. If the outcome measure diverges in the treatment period, then the treatment is assumed to have caused the difference. If the outcome measures for the treated group and synthesized control group do not diverge, then it can be assumed that the treatment did not affect the outcome. Finally, the synthesized control group's outcome measure should match the treated unit's outcome measure during the pretreatment period.

The advantages of using an SCM procedure over a fixed effects procedure or a case study method are numerous. First, a weighted combination of control states provides a much better comparison to the treated state than a single control state (Abadie et al., 2010). Second, the relative contribution of each state in the synthetic control group can be ascertained (Abadie et al., 2010). Third, in an SCM procedure, it can be determined if there are differences with regards to the intervention variable and the other predictor variables between the treated state and the control group (Abadie et al., 2010). Due to these advantages, the SCM statistical procedure has been used in several studies examining the effects of public policies, laws, and exogenous shocks on various outcome measures (Kreif et al., 2016; Abadie et al., 2015, 2010; Abadie and Gardeazabal, 2003).

For purposes of the present study, the "treatment" is considered to be when a state changes its CCW laws from "prohibited" to "shall issue". Hence, only those states that changed their CCW laws from "prohibited" to "shall issue" are in the treatment group. All other states are in the potential control group. From this set of possible controls, a synthesized control group was created.

The outcome variables are the gun related murder rate (gun-related murders per 100,000 persons) and the murder rate (murders per 100,000 persons). The primary reason murder rates, instead of other crime rates, were used is because it is important to differentiate between total murders and gun-related murders. Given that the enactment of a permissive CCW law may reduce gun-related murders, it is imperative that this type of outcome measure is examined. In addition, state-level data on the incidence of other types of gun related crimes are either non-existent or are unreliable.

The predictor variables used in this analysis were selected based upon their use in prior research (Barati, 2016; Bartley and Cohen, 1998; Gius, 2014; Lott and Mustard, 1997; Moody and Marvell, 2009; Moody, 2001; Olson and Maltz, 2001). These variables include the percentage of the state population that is African-American, per capita real income, percentage of population that is college educated, unemployment rate, percentages of population aged 18 to 24 and 25 to 34, population density, per capita alcohol consumption, the ratio of gun-related suicides to total suicides, and the percentage of the state's population that lives in large cities. The statistical software package R was used to conduct the SCM (Abadie et al., 2011). States that had missing observations were excluded from the analyses. Eight states were in the treatment group, and 31 states were in the potential control group.

Only one other study used the synthetic control method to examine the impact of a change in CCW laws on crime rates (Donohue et al., 2017). The present study differs from this prior research in that the present study examines murder rates and defines the treatment as a change in CCW laws from "prohibited" to "shall issue". Donohue et al. (2017) looked at violent crime rates,

and they only examined the repeal of the prohibition of concealed carry.

In order to test the robustness of the results obtained from the SCM analysis, a fixed effects model that controls for both state-level and year fixed effects was also estimated. All observations were weighted using state-level population (to correct for potential heteroscedasticity), standard errors were corrected using a clustering method (standard errors were clustered at the state level), and a log-linear functional form was used. Given the above, the following equation was estimated in the present study:

$$\ln Y_{i,t} = \alpha_0 + \alpha_i + \gamma_t + \beta'X + \varepsilon_{i,t} \quad (1)$$

In the above equation, Y denotes the murder rate or gun-related murder rate, α_i denotes the state-level effects, γ_t denotes the year effects, and X denotes the vector of explanatory variables which includes a concealed carry dummy variable that denotes a change from "prohibited" status to "shall issue" status. This model is very similar to those used by other studies on this topic (Barati, 2016; Bartley and Cohen, 1998; Gius, 2014; Lott and Mustard, 1997; Moody and Marvell, 2009; Moody, 2001; Olson and Maltz, 2001).

In order to determine if fixed or random effects was more appropriate in the present study, a Hausman Test was used. Results of the test suggested that fixed effects was the more appropriate model. Clustering standard errors was necessary in order to account for potentially nonrandom variations within certain groups. Although there has been some criticism regarding the use of clustering to correct standard errors, most of this criticism was directed at studies that used too few clusters (Cameron et al., 2008). The present study uses many state-level clusters, thus justifying the use of clustered standard errors.

A log-linear function was used because it corrects for nonlinearities in the data. In order to test the appropriateness of the log-linear functional form, both the extended projection test for non-nested hypotheses (Davidson and MacKinnon, 1981) and the Ramsey test (Ramsey, 1969) were used. Both tests indicated that the log-linear model was more appropriate in the present study.

It is important to note that, given that the functional form used in the present study is log-linear, the coefficients on the dummy variables must be transformed in order to be properly interpreted. For example, if the coefficient on a dummy variable is 0.2, then that variable is associated with a 22% increase in the dependent variable being examined ($e^{0.2} = 1.22$ or 22%).

In order to determine the effect of a change in CCW status from "prohibited" to "shall issue", a CCW dummy variable was created that takes the value of one if the state changed their gun control laws from "prohibited" to "shall issue"; otherwise, the CCW dummy variable takes the value of zero. A state that changes from "prohibited" to "shall issue" will only have a value of one for the CCW dummy variable when the state has a "shall issue" CCW law. Although there has been some criticism in the past regarding the use of binary variables to denote the status of gun control in a particular state, almost all prior studies use dummy variables in their regression analyses. Some of the studies that used this approach are Gius (2014), Rubin and Dezhbakhsh (2003), Dezhbakhsh and Rubin (1998), Lott and Mustard (1997), and Kleck and Patterson (1993).

Another issue with using a binary variable to denote the status of gun control laws in a particular state is that a change in gun control laws may be coincident with an exogenous change in the crime rate, thus possibly resulting in a spurious correlation between gun control and crime (Moody, 2001). One way to test for this possibility is to include as explanatory variables in the crime regression both lags and leads of the CCW dummy variable. If these variables are insignificant, then this potential source of error does not exist (Moody, 2001). In the present study, this error was tested for by including two lags and two leads of the CCW dummy variable.

Results of this test indicated that this type of spurious correlation did not exist. Results of this test are available upon request.

In addition to concealed carry laws, it is also assumed that murder rates are dependent upon state demographics and various other state-level socioeconomic factors. The same explanatory variables that were used in the SCM analysis are used in the fixed effects regressions. As noted earlier, these variables were selected based upon their use in prior research (Barati, 2016; Bartley and Cohen, 1998; Gius, 2014; Lott and Mustard, 1997; Moody and Marvell, 2009; Moody, 2001; Olson and Maltz, 2001).

State-level data on murder and gun-related murder rates were obtained from the *Supplementary Homicide Reports* (1990–2014), which were provided by the Bureau of Justice Statistics, U.S. Department of Justice. Unfortunately, some of the values for murders committed were suspiciously low. In order to maintain the integrity of the data, any suspicious observations were deleted. The method by which suspicious data were identified was through a linear trend of the gun murder rates at the state level. If a data point was identified as an outlier, then that observation was deleted. Using this methodology, only 18 observations were deleted from the data set. Given that the final data set used in the fixed effects analysis had 1203 observations, these deletions were not expected to have any appreciable effect on the results of the fixed effects analysis. In addition, there were 29 missing observations (murder rates) in the original SHR data set. Hence, there were a grand total of 47 missing observations in the data used in the fixed effects regression.

It is important to note that, in the fixed effects regressions, the existence of missing observations did not require that the entire state be eliminated from the data set. Only that year's data in which there was a missing observation had to be deleted. Hence, for the fixed effects models, an unbalanced panel data set was used to estimate the regressions. In the SCM analysis, however, states had to have data for every year examined (1990–2014); no missing observations were allowed in the data. Hence, eleven states were eliminated from the original data in order to construct a data set that could be used in the SCM analysis.

Information on CCW laws were obtained from Gottlieb (1991), Gottlieb (1981), Henderson (2005), Henderson, (2000), Ludwig and Cook (2003), and the National Rifle Association. If the above references contradicted one another, then the author examined the original state law in order to determine the status of the CCW law in a particular state. State-level data on total suicides and firearm-related suicides were obtained from the National Center for Injury Prevention and Control, the Centers for Disease Control (CDC). The WISQARS system is used to obtain the necessary data from the CDC website. Per capita alcohol consumption data were obtained from the National Institute on Alcohol Abuse and Alcoholism. All other state-level data were obtained from relevant Census Bureau reports. Data used in the present study is for the years 1990–2014. The sample size for the fixed effects model is 1203. Data used for the SCM analyses differ somewhat from the fixed effects model data in that there is only one treated state per SCM estimation. Hence, the sample sizes of the eight data sets used for the SCM analyses were 800. Descriptive statistics for the fixed effects data set are presented on Table 2.

3. Results

For the SCM analysis, the eight states that were in the treatment group (changed from “prohibited” CCW to “shall issue”) are as follows: Arkansas (1995), Missouri (2003), Nebraska (2006), New Mexico (2003), North Carolina (1995), Ohio (2004), Oklahoma (1995), and Texas (1995). Dates noted in parentheses are when the states adopted “shall issue” CCW laws. Although several other states also switched from “prohibited” to “shall issue”, these states

Table 2

Descriptive Statistics (Fixed Effects Data).

Variable	Mean	Standard Deviation
Gun-related murder rate (per 100,000 persons)	3.15	2.21
Murder rate (per 100,000 persons)	5.13	2.93
Percent of population that is African-American	0.099	0.095
Per capita real income	\$16,413	\$2,999
Percent of population college educated	0.25	0.06
Unemployment rate	0.057	0.019
Gun-related suicide ratio	0.56	0.13
Percent population 18–24	0.099	0.009
Percent population 25–34	0.14	0.016
Population density	183.83	251.95
Per capita alcohol consumption	2.32	0.49
Percent population in large cities	0.137	0.137

were not included due to missing observations and incomplete data.

Although several states enacted “unrestricted” CCW laws (no permit required for concealed carry) in the past several years, very few states adopted such laws prior to 2014. Hence, this lack of data precluded any analysis pertaining to the impact of “unrestricted” concealed carry on murder rates.

An important aspect of the SCM analysis is that the actual outcome measure for a treated state should be similar to the outcome measure for the synthetic version of the treated state in the pre-treatment period. In order to test the similarity between the outcome measures for the treated state and the synthetic state, two statistical measures were used. The first measure was the Mean Squared Prediction Error (MSPE) in the pre-treatment period. The larger the MSPE, the greater is the difference between the two measures. Unfortunately, the MSPE is affected by the units of measurement and the scale of the outcome measure.

The other statistical test that was used is the hypothesis test for the difference between the means of the actual outcome measure and the synthetic outcome measure. In the pre-treatment period, this test should be statistically insignificant, thus indicating that there is no statistical difference between the actual outcome measure and the synthetic outcome measure. Both the MSPE and the pre-treatment period *t*-tests are presented on Table 3. As can be seen from these results, several states have *t*-tests that are statistically significant in the pre-treatment period, thus indicating that the actual outcome measure differs from the synthetic outcome measure; these states were excluded from the SCM analysis. After excluding states that had significant *t*-tests in the pre-treatment period, there were only four states that remained in the treated state pool.

In order to determine if the change in CCW law significantly affected the murder rates in the post-treatment period, the hypothesis test for the difference between the means of the actual outcome measure and the synthetic outcome measure was once again employed. In the post-treatment period, if the law had a significant effect on the murder rates, then the *t*-test should be statistically significant. These results are also reported on Table 3.

The results of the SCM analyses on the four treated states are presented on Chart 1. The vertical line on the chart denotes the year when a “shall issue” law was adopted. As can be seen from these charts and from the post-treatment *t*-tests provided on Table 3, the only state that experienced a consistent divergence in the outcome variable post-treatment was New Mexico (murder and gun-related murder). The switch from “prohibited” CCW to “shall issue” CCW resulted in an increase in both total murder rates and gun related murder rates in New Mexico.

For all other treated states, the switch from “prohibited” to “shall issue” had no statistically significant effects on either gun-related murder rates or total murder rates. Hence, according to the SCM results, the overall impact of a loosening of concealed carry laws on

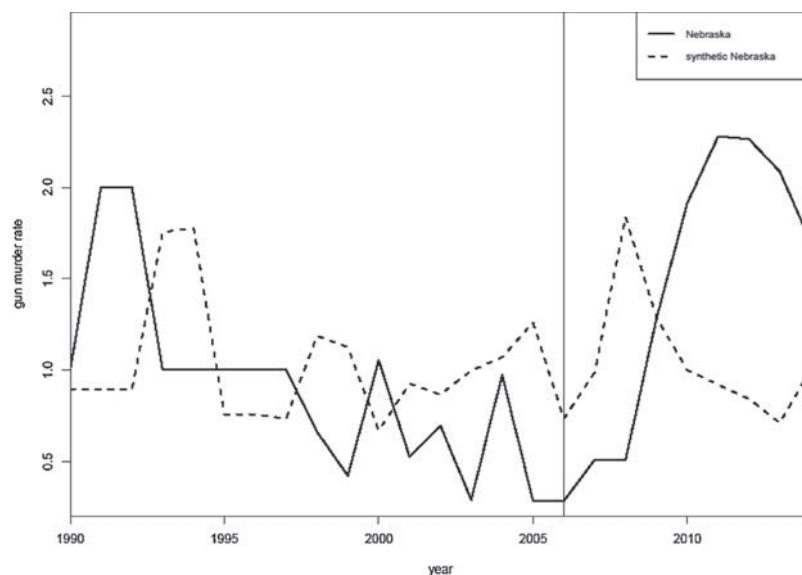
Table 3Treatment States MSPE and *t*-tests for Pre- and Post-Treatment Periods.

State	MSPE	Pre-Treatment <i>t</i> Test Statistic	Post-Treatment <i>t</i> Test Statistic
Arkansas (gun murder) ^a	0.965	1.603*	–1.413
Arkansas (murder) ^a	3.115	3.33***	0.0557
Missouri (gun murder) ^a	1.195	2.128**	4.514***
Missouri (murder) ^a	2.719	2.6711***	5.369***
Nebraska (gun murder)	0.400	–0.694	1.33
Nebraska (murder)	1.166	–0.1514	–1.304
New Mexico (gun murder)	0.542	0.295	2.816***
New Mexico (murder)	2.184	–0.0129	2.299**
North Carolina (gun murder) ¹	0.335	2.091**	0.381
North Carolina (murder) ^a	1.386	3.347***	–0.436
Ohio (gun murder) ^a	1.445	–3.548***	–3.028***
Ohio (murder)	0.4045	–0.0297	–0.767
Oklahoma (gun murder)	0.1026	0.0148	0.1916
Oklahoma (murder)	0.469	0.0110	1.33
Texas (gun murder) ^a	2.595	2.133**	–1.011
Texas (murder) ^a	8.885	3.3675***	–2.558***

Notes.

MSPE denotes the Mean Squared Prediction Error for the pre-treatment period.

*10% significance; **5% significance; ***1% significance.

^a The synthetic outcome was statistically significantly different from the actual outcome in the pre-treatment period. States that possess this attribute were not included in the SCM analysis.**Chart 1.** Nebraska SCM Results CCW Law Had No Effect on Gun-Related Murder Rate (Vertical line denotes year CCW law was changed.).

Weights Given to Control States.

Connecticut = 2.2%.

South Dakota = 84%.

Virginia = 13.8%.

All Others = 0%.

murder rates was somewhat mixed and inconclusive. These results are not directly comparable to those found in *Donohue et al. (2017)* primarily because their study examined the effects of CCW laws on violent crime rates and not on murder rates. Nonetheless, *Donohue et al. (2017)* found that the repeal of the prohibition of concealed carry resulted in an increase in violent crime rates.

In order to test the robustness of the synthetic control model in this scenario, two placebo tests were conducted (*Abadie et al., 2011*). In this test, the SCM is applied to a control state that is similar to the treated state but did not enact a change in its CCW law. It is assumed that the change in the similar control state occurred in the same year as that in the treated state. The only treated state tested in this manner was New Mexico. For gun related murders, the most similar state was Arizona, and for murder rates, the most similar state was Mississippi. Results are presented on *Charts 8 and 9*. As

can be seen from these charts, the outcome trajectories for Arizona and Mississippi and their synthetic counterparts are very similar.

Another method that was used to test the robustness of the SCM results was a permutation test (*Abadie et al., 2011*). In this test, the control states are subjected to the same synthetic control method as was the treated state. Then, the gaps between the synthetic outcomes and the actual outcomes for each of the control states and the treated state (New Mexico) are then plotted on a chart in order to determine if the true synthetic control unit is different from the other control units. Ideally, there should be small gaps prior to the treatment and large gaps afterwards. Results are presented on *Charts 10 and 11*. Please note that in New Mexico (the treated state), the change in CCW laws occurred in 2003. As can be seen from these charts, the outcome measures track very similarly pre-treatment, but after 2003, there is much more of a divergence. This denotes that the change in murder and gun related murder rates

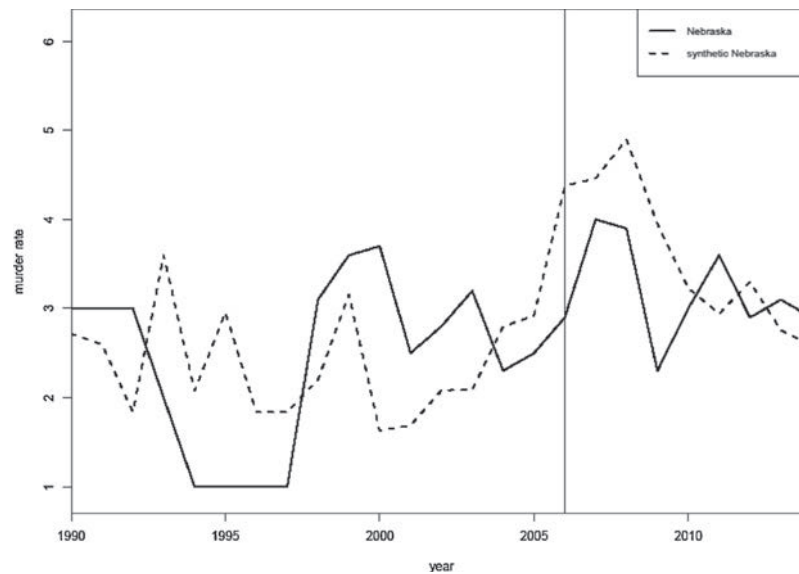


Chart 2. Nebraska SCM Results CCW Law Had No Effect on Murder Rate (Vertical line denotes year CCW law was changed.).

Weights Given to Control States.

Arizona = 12%.

South Dakota = 88%.

All Others = 0%.

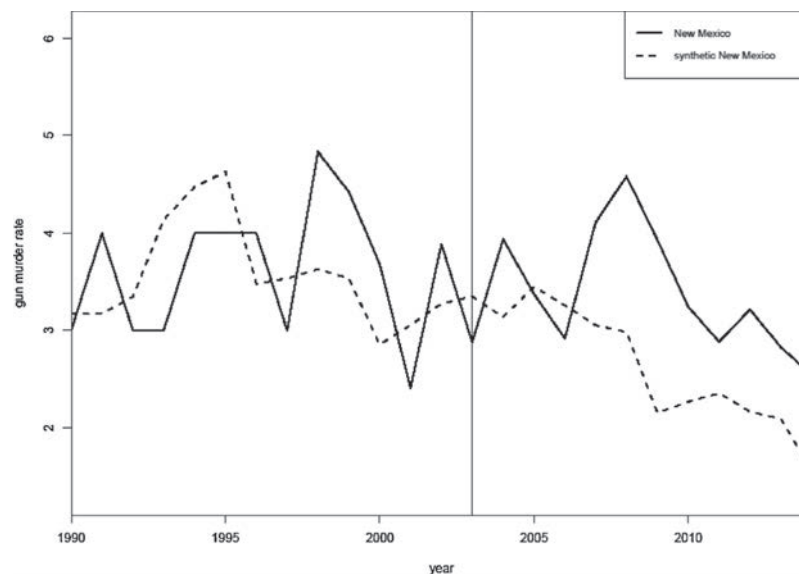


Chart 3. New Mexico SCM Results CCW Law Significantly Increased Gun-Related Murder Rate (Vertical line denotes year CCW law was changed.).

Weights Given to Control States.

Arizona = 47.6% West Virginia = 15%.

Nevada = 3.3% All Others = 0%.

North Dakota = 5.8%.

South Dakota = 28.3%.

cannot be attributed to the change in CCW laws in the control states (Abadie et al., 2011).

In the final test on the robustness of the results obtained from the SCM analysis, a fixed effects model that controls for both state-level and year fixed effects was also estimated. Observations were weighted using state-level population, standard errors were corrected using a clustering method, and a log-linear functional form was used. Results are presented on Tables 4 and 5.

These results suggest that states that switched from “prohibited” CCW status to “shall issue” CCW status experienced an increase in both the gun-related murder rate and the total murder rate. According to these results, states that switched from “pro-

hibited” to “shall issue” had gun-related murder rates that were 12.3% higher than other states, and they had total murder rates that were 4.9% higher than other states. These results differ from those found in Barati (2016) who found that a change from “prohibited” CCW status to “shall issue” had no statistically significant effects on murder rates.

4. Conclusion

Even though murder rates are lower now than they have been in decades, many individuals still believe that guns are needed for self-defense, even in public settings. These individuals believe that

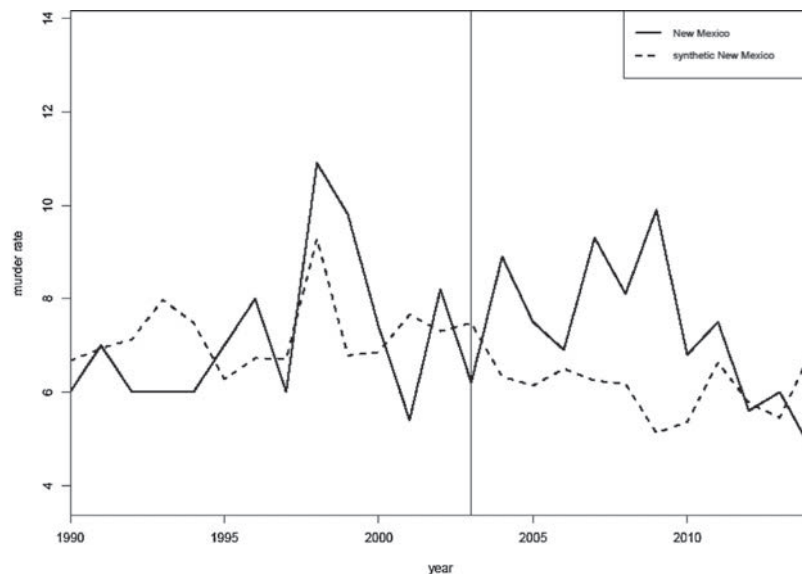


Chart 4. New Mexico SCM Results CCW Law Significantly Increased Murder Rate (Vertical line denotes year CCW law was changed.).

Weights Given to Control States.

Alaska = 14.5% All Others = 0%.

Mississippi = 60.5%.

Nevada = 5%.

Wyoming = 20%.

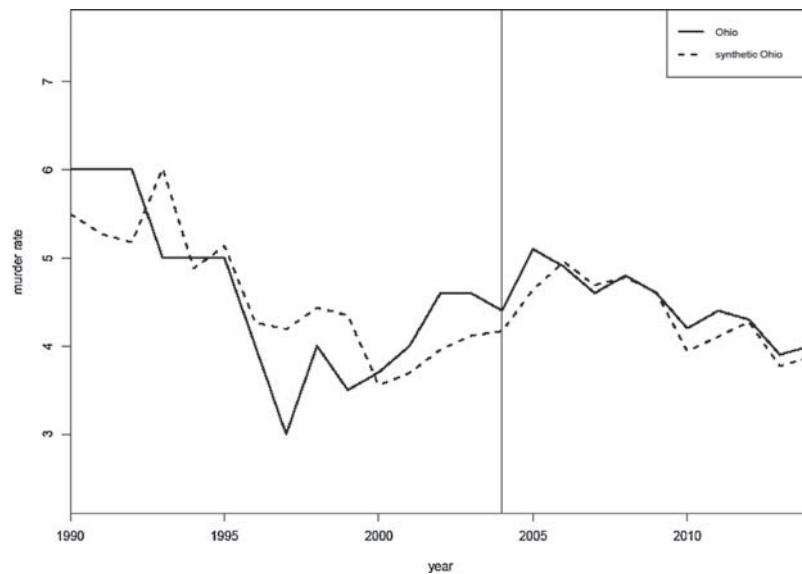


Chart 5. Ohio SCM Results CCW Law Had No Effect on Murder Rate (Vertical line denotes year CCW law was changed.).

Weights Given to Control States.

Indiana = 1.2% Maryland = 1.9% Pennsylvania = 16.2%.

Hawaii = 1.1% Rhode Island = 1.2% West Virginia = 17.4%.

New Jersey = 13.9% South Dakota = 23.3% Tennessee = 11.6%.

All Others = 12.2%.

allowing private citizens to carry guns would deter potential criminals from committing criminal acts. Unfortunately, much of the evidence to date suggests that permissive CCW laws have little to no effect on crime. Nonetheless, many states over the past twenty years have loosened regulations regarding the concealed carry of firearms.

Although there have been numerous studies examining CCW laws since Lott and Mustard (1997), the present study takes a different approach in examining the effects of CCW laws on crime. First, the present study looks at those states that changed their CCW laws from “prohibited” to “shall issue”. The only other study

that specifically examined this type of shift in the legal status of concealed weapons was Barati (2016). Second, the present study is the only published study that uses the synthetic control method to determine the effects of a change in CCW laws on murder rates. As noted earlier, most prior studies that examined the effects of CCW laws on crime either used a fixed effects model or a difference-in-differences model.

Using SCM, it was found that only one of the four treated states experienced an increase in murder rates when they switched from “prohibited” CCW status to “shall issue” status.

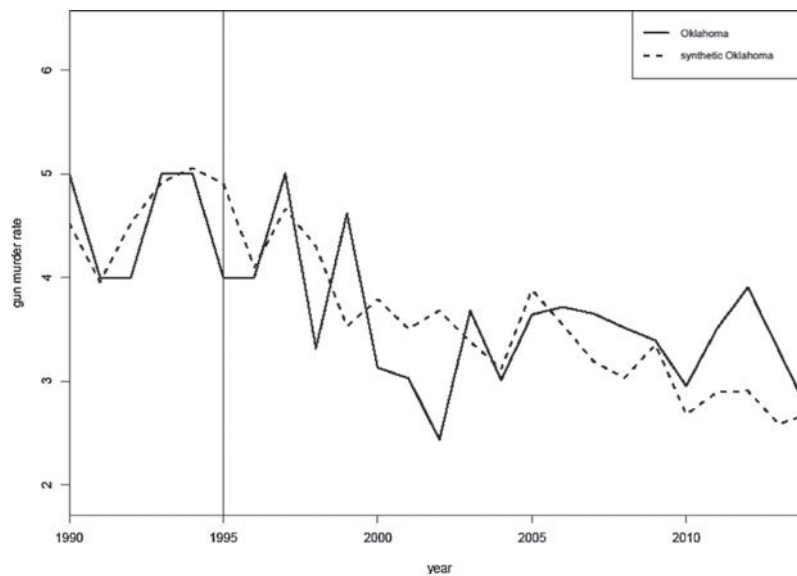


Chart 6. Oklahoma SCM Results CCW Law Had No Effect on Gun Related Murder Rate (Vertical line denotes year CCW law was changed.).

Weights Given to Control States.

Arizona = 10%.

Oregon = 29.6%.

Tennessee = 56.5%.

Utah = 3.9%.

All Others = 0%.

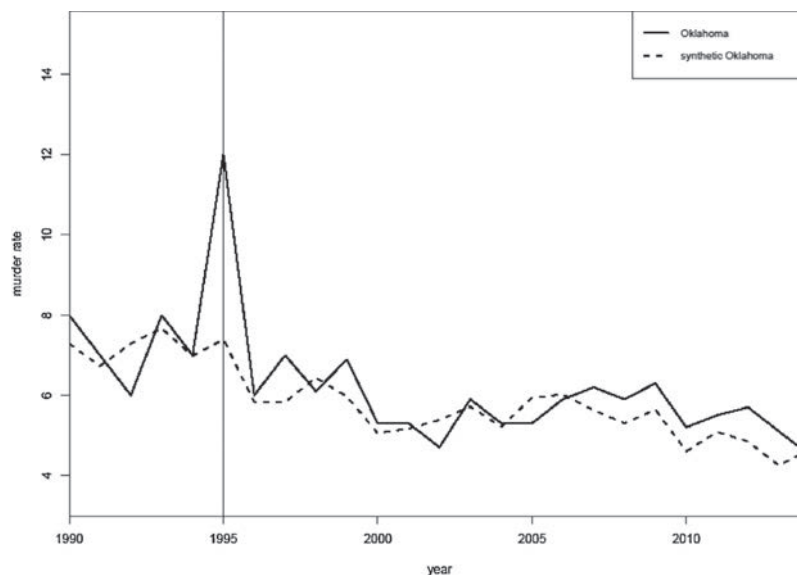


Chart 7. Oklahoma SCM Results CCW Law Had No Effect on Murder Rate (Vertical line denotes year CCW law was changed.).

Weights Given to Control States.

Arizona = 21.4% All Others = 0%.

Tennessee = 34.5%.

Utah = 5.7%.

West Virginia = 38.3%.

As a check on the robustness of the SCM results, a two-way fixed effects model was also estimated, and it was found that states that switched from “prohibited” to “shall issue” experienced a 12.3% increase in gun-related murder rates and a 4.9% increase in overall murder rates when compared to other states. These results hold even after controlling for important demographic variables such as the percentage of state’s population that is African-American, gun ownership (ratio of firearm suicides to total suicides), and alcohol consumption.

It is important to note that none of the results of the present study suggest that a move from “prohibited” CCW status to “shall issue” CCW is associated with a decline in murder rates. Hence, the deterrent effects of permissive CCW laws that have been discussed at great lengths in many prior studies do not appear to be supported by the evidence found in this study (Kovandzic and Marvell, 2003; Kovandzic et al., 2005; Lott and Mustard, 1997).

Although the fixed effects results suggest that a switch from “prohibited” CCW status to “shall issue” status increases murder rates on average, the results obtained from the SCM methodology

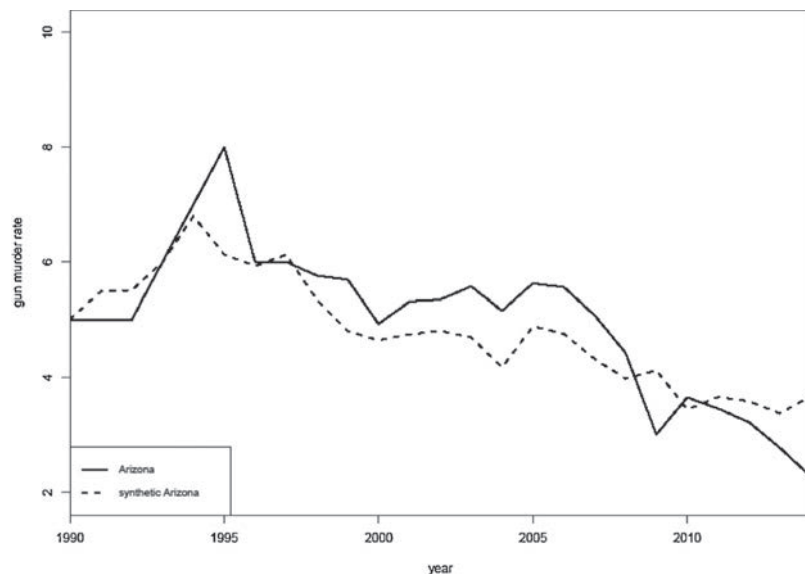


Chart 8. Placebo Test: Trends in Gun Murder Rates: Arizona vs. synthetic Arizona.

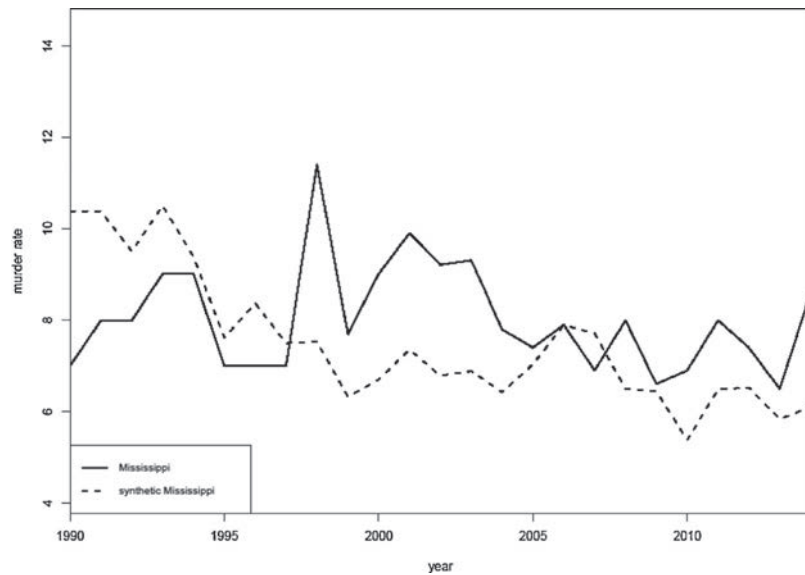


Chart 9. Placebo Test: Trends in Murder Rates: Mississippi vs. synthetic Mississippi.

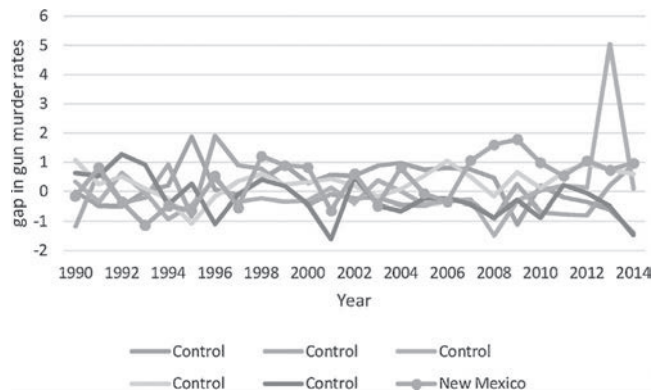


Chart 10. Permutation Test: Gun Murder Rates in New Mexico and Control States. Control States: Arizona, West Virginia, Nevada, North Dakota, and South Dakota.

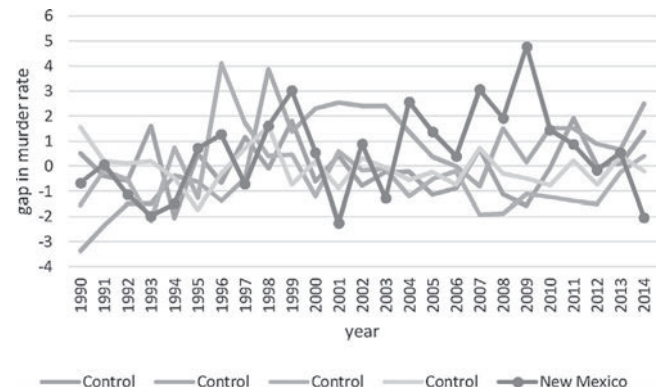


Chart 11. Permutation Test: Murder Rates in New Mexico and Control States. Control States: Alaska, Mississippi, Nevada, and Wyoming.

Table 4

Fixed Effects – Gun-Related Murder Rate.

Variable	Coefficient	Test Statistic
Constant	−0.81	−1.78*
CCW dummy variable	0.116	3.21***>
Percent of population that is African-American	7.76	5.67***>
Per capita real income	−0.000016	−1.39
Percent of population college educated	−0.147	−0.49
Unemployment rate	0.753	0.85
Gun-related suicide ratio	0.92	2.96***>
Percent population 18–24	−2.12	−2.30**
Percent population 25–34	−2.42	−1.66*
Population density	0.0023	3.20***>
Per capita alcohol consumption	0.467	5.99***>
Percent population in large cities	−0.93	−1.63

R² = 0.87.

1% p-value ***>; 5% p-value **; 10% p-value *.

Table 5

Fixed Effects – Murder Rate.

Variable	Coefficient	Test Statistic
Constant	0.167	0.48
CCW dummy variable	0.048	1.74*
Percent of population that is African-American	6.71	6.44***>
Per capita real income	−0.000019	−2.24**
Percent of population college educated	−0.0492	−0.21
Unemployment rate	1.26	1.87*
Gun-related suicide ratio	0.548	2.31**
Percent population 18–24	−1.33	−1.90*
Percent population 25–34	−0.21	−0.19
Population density	−0.000015	−0.03
Per capita alcohol consumption	0.457	7.69***>
Percent population in large cities	−0.898	−2.06**

R² = 0.871.

1% p-value ***>; 5% p-value **; 10% p-value *.

indicate that these effects may be more state-specific and may not be applicable to all states. In addition, the fixed effects methodology may not be the most appropriate statistical technique to use to evaluate the effects of CCW laws on crime rates. Donohue et al. (2017) note that “the problems posed by high-dimensional estimation, misspecified models, and a lack of knowledge of the correct set of explanatory variables” may pose insurmountable problems when attempting to estimate the effects of any gun control legislation on crime (Donohue et al., 2017, p.16).

Another potential problem with the results found in the present study is the uncertainty involved with any examination of the effects of concealed carry laws on crime over the past 30 years. Crime rates increased during the 1980s and early 1990s and then fell to historic lows. During this period, however, there were numerous changes in gun control laws, both at the state and Federal level. Most of the changes loosened existing statutes, but some laws increased restrictions on various types of firearms. Hence, there were changes in the regulation of firearms at the same time that there were societal changes that were resulting in lower crime rates. Donohue (2003) believes that “... a disaggregated data push toward a more-guns, more-crime conclusion. ...” (pg. 324) and that “... crime swings that occurred in the late 1980s and 1990s happened to correlate with the passage of shall-issue laws, and the panel data model seems unable to separate out the contribution of the relatively minor influence of the shall issue law from the major impacts of these broad swings” (Donohue, 2003, pg. 325). Therefore, even though some of the results of the present study suggest that concealed carry laws are significantly and positively related to murder, it is reasonable to assume that various societal factors may have much greater effects on crime than any single gun control statute.

Finally, given the inconclusiveness of the SCM results, no public policy proposals should be gleaned from this study. Some prior studies have shown that crime rates are negatively affected by permissive CCW laws, while other studies have found that crime rates are either unaffected or are positively related to CCW laws. This inconclusiveness is no doubt due to the variety of CCW laws at the state level and the uncertainty regarding the frequency with which citizens avail themselves of the opportunity to defend themselves from criminal acts. Given that one of the goals of CCW laws is deterrence, it is difficult to measure deterrence if it is unknown how many persons are actually carrying concealed weapons on a regular basis. Binary variables denoting the legal status of concealed carry laws are poor substitutes for this type of data.

References

- Abadie, A., Diamond, A., Hainmueller, J., 2015. Comparative politics and the synthetic control method. *Am. J. Pol. Sci.* 59 (2), 495–510.
- Abadie, A., Diamond, A., Hainmueller, J., 2011. Synth: an r package for synthetic control methods in comparative case studies. *J. Stat. Softw.* 42 (13), 1–17.
- Abadie, A., Diamond, A., Hainmueller, J., 2010. Synthetic control methods for comparative case studies: estimating the effect of California's tobacco control program. *J. Am. Stat. Assoc.* 105 (490), 493–505.
- Abadie, A., Gardeazabal, J., 2003. The economic costs of conflict: a case study of the basque country. *Am. Econ. Rev.* 93 (1), 113–132.
- Ayers, Ian, Donohue, John, 2003. Shooting down the more guns, less crime hypothesis. *Stanford Law Rev.* 55, 1193–1314.
- Barati, M., 2016. New evidence on the impact of concealed carry weapon laws on crime. *Int. Rev. Law Econ.* 47, 1–8.
- Bartley, William, Cohen, Mark, 1998. The effect of concealed weapons laws: an extreme bound analysis. *Econ. Inq.* 36, 258–265.
- Benson, Bruce, Mast, Brent, 2001. Privately produced general deterrence. *J. Law Econ.* 44 (S2), 725–746.
- Black, Dan, Nagin, Daniel, 1998. Do Right-To-Carry Laws Deter Violent Crime? *J. Legal Stud.* 27, 209–219.
- Blau, Benjamin, Gorry, Devon, Wade, Chip, 2016. Guns, laws, and public shootings in the United States. *Appl. Econ.* 48 (49), 4732–4746.
- Bronars, Stephen, Lott, John, 1998. Criminal deterrence, geographic spillovers, and the right to carry concealed handguns. *Am. Econ. Rev.* 88 (2), 475–479.
- Cameron, C., Gelbach, J., Miller, D., 2008. Bootstrap-based improvements for inference with clustered errors. *Rev. Econ. Stat.* 90 (3), 414–427.
- Crifasi, Cassandra, Pollack, Keshia, Webster, Daniel, 2016. Effects of state-level policy changes on homicide and nonfatal shootings of law enforcement officers. *Inj. Prev.* 22, 274–278.
- Davidson, R., MacKinnon, J., 1981. Several tests for model specification in the presence of alternative hypotheses. *Econometrica* 49, 781–793.
- Devaraj, Srikant, Patel, Pankaj, 2018. An examination of the effects of 2014 concealed weapons law in Illinois on property crimes in Chicago. *Appl. Econ. Lett.* 25 (16), 1125–1129.
- Dezhbakhsh, Hashem, Rubin, Paul, 1998. Lives saved or lives lost? The effects of concealed handgun laws on crime. *Am. Econ. Rev.* 88 (2), 468–474.
- Donohue, John, 2003. The impact of concealed-carry laws. In: Ludwig, John, Cook, Philip (Eds.), *Evaluating Gun Policy: Effects on Crime and Violence*. The Brookings Institution, Washington, D.C., pp. 287–341.
- Donohue, John, Aneja, Abhay, Weber, Kyle, 2017. “Right-to-Carry Laws and Violent Crime: A Comprehensive Assessment Using Panel Data and a State-level Synthetic Controls Analysis.” NBER Working Paper.
- Duwe, Grant, Kovandzic, Tomislav, Moody, Carlisle, 2002. The impact of right-to-Carry concealed firearm laws on mass public shootings. *Homicide Stud.* 6 (4), 271–296.
- Ginwalla, Rashna, Rhee, Peter, Friese, Randall, Green, Donald, Gries, Lynn, Joseph, Bellal, Kulvatunyou, Narong, Lubin, Dafney, O'Keeffe, Terence, Vercruysse, Gary, Wynne, Julie, Tang, Andrew, 2013. Repeal of the concealed weapons law and its impact on gun-related injuries and deaths. *J. Trauma Acute Care Surg.* 76 (3), 569–575.
- Gius, Mark, 2014. An examination of the effects of concealed weapons laws and assault weapons bans on state-level murder rates. *Appl. Econ. Lett.* 21 (4), 265–267.
- Gottlieb, Alan, 1981. *The Rights of Gun Owners*. Caroline House Publishers, Aurora, Illinois.
- Gottlieb, Alan, 1991. *The Rights of Gun Owners*. Merril Press, Bellevue, Washington.
- Helland, Eric, Tabarrok, Alexander, 2004. Using placebo laws to test “More guns, less crime. *Advances in Economic Analysis & Policy* 4 (1), 1–7.
- Henderson, Harry, 2000. *Gun Control. Facts on File*, New York, New York.
- Henderson, Harry, 2005. *Gun Control. Facts on File*, New York, New York.
- Kleck, Gary, Patterson, E. Britt, 1993. The impact of gun control and gun ownership levels on violence rates. *J. Quant. Criminol.* 9 (3), 249–287.
- Kovandzic, Tomislav, Marvell, Thomas, 2003. Right-to-Carry Concealed Handguns and Violent Crime: Crime Control through Gun Decontrol? *Criminol. Public Policy* 2 (3), 363–396.

- Kovandzic, Tomislav, Marvell, Thomas, Vieraitis, Lynne, 2005. The Impact of “Shall Issue” Concealed Handgun Laws on Violent Crime Rates: Evidence from Panel Data for Large Urban Cities. *Homicide Stud.* 9 (4), 292–323.
- Kreif, Noemi, Grieve, Richard, Hangartner, Dominik, James, Alex, Turner, Silviya Nikolova, Sutton, Matt, 2016. Examination of the synthetic control method for evaluating health policies with multiple treated units. *Health Econ.* 25, 1514–1528.
- Lott, John, 1998. The concealed-handgun debate. *J. Legal Stud.* 27 (1), 221–243.
- Lott, John, Mustard, David, 1997. Crime, deterrence, and right-to-Carry concealed handguns. *J. Legal Stud.* 26 (1), 1–68.
- Ludwig, Jens, 1998. ‘Concealed-Gun-Carrying laws and violent crime: evidence from state panel data. *Int. Rev. Law Econ.* 18 (3), 239–254.
- Ludwig, John, Cook, Philip (Eds.), 2003. *Evaluating Gun Policy: Effects on Crime and Violence*. The Brookings Institution, Washington, D.C.
- Moody, Carlisle, 2001. Testing for the effects of concealed weapons laws: specification errors and robustness. *J. Law Econ.* 44 (S2), 799–813.
- Moody, Carlisle, Marvell, Thomas, 2009. The Debate on Shall Issue Laws, Continued. *Econ J. Watch* 6 (2), 203–217.
- Moody, Carlisle, Marvell, Thomas, Zimmerman, Paul, Alemante, Fasil, 2014. The impact of right-to-Carry laws on crime: an exercise in replication. *Q. Rev. Econ. Finance* 4 (1), 33–43.
- Olson, David, Maltz, Michael, 2001. Right-to-Carry concealed weapon laws and homicide in large U.S. counties: the effect on weapon types, victim characteristics, and victim-offender relationships. *J. Law Econ.* 44 (S2), 747–770.
- Phillips, Charles, Nwaiwu, Obioma, Lin, Szu-hsuan, Edwards, Rachel, Imanpour, Sara, Ohsfeldt, Robert, 2015. Concealed handgun licensing and crime in four states. *Isrn Discret. Math.*, 803742.
- Plassman, Florenz, Tideman, T. Nicolaus, 2001. Does the Right to Carry Concealed Handguns Deter Countable Crimes? Only a Count Analysis Can Say. *J. Law Econ.* 44 (2), 771–798.
- Ramsey, J.B., 1969. Tests for specification errors in classical linear least-squares regression analysis. *J. R. Stat. Soc. Ser. B* 31, 350–371.
- Rubin, Paul, Dezhbakhsh, Hashem, 2003. The effect of concealed handgun laws on crime: beyond the dummy variables. *Int. Rev. Law Econ.* 23, 199–216.
- Shi, Wei, Lee, Lung-fei, 2017. The effects of gun control on crimes: a spatial interactive fixed effects approach. *Empir. Econ.*, <http://dx.doi.org/10.1007/s00181-017-1415-2>.
- Smith, Michael, Petrocelli, Matthew, 2018. The effect of concealed handgun carry deregulation in Arizona on crime in Tucson. *Crim. Justice Policy Rev.*, 1–18.

EXHIBIT 7



Legal status and source of offenders' firearms in states with the least stringent criteria for gun ownership

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ABSTRACT

Background Gun possession by high-risk individuals presents a serious threat to public safety. U.S. federal law establishes minimum criteria for legal purchase and possession of firearms; many states have laws disqualifying additional categories for illegal possession.

Methods We used data from a national survey of state prison inmates to calculate: 1) the proportion of offenders, incarcerated for crimes committed with firearms in 13 states with the least restrictive firearm purchase and possession laws, who would have been prohibited if their states had stricter gun laws; and 2) the source of gun acquisition for offenders who were and were not legally permitted to purchase and possess firearms.

Results Nearly three of ten gun offenders (73 of 253 or 28.9%) were legal gun possessors but would have been prohibited from purchasing or possessing firearms when committing their most recent offense if their states had stricter prohibitions. Offenders who were already prohibited under current law acquired their gun from a licensed dealer, where a background check is required, five times less often than offenders who were not prohibited (3.9% vs. 19.9%; $\chi^2=13.31$; $p\leq 0.001$). Nearly all (96.1%) offenders who were legally prohibited, acquired their gun from a supplier not required to conduct a background check.

Conclusions Stricter gun ownership laws would have made firearm possession illegal for many state prison inmates who used a gun to commit a crime. Requiring all gun sales to be subject to a background check would make it more difficult for these offenders to obtain guns.

INTRODUCTION

Gun violence has long been one of the most significant public safety and social problems in the USA. In the USA, in 2008, gun violence resulted in 12 179 homicides and an estimated 56 626 assaultive injuries serious enough to warrant a hospital emergency room visit.¹ Among high-income countries, the USA is unique in its extraordinarily high rate of homicides. This disparity is most striking for homicides committed with firearms where the US rate is 20 times higher than other high-income countries.²

Despite the magnitude of the problem, US gun policy rarely considers appropriate criteria for disqualifying someone from lawfully possessing a firearm. Federal law disqualifies certain groups of high-risk individuals from owning guns, including felons, fugitives, unlawful users of or those addicted to controlled substances, those who have been 'adjudicated as a mental defective' or committed to a mental institution, individuals who

have been dishonourably discharged from the armed forces, persons subject to certain domestic violence restraining orders, persons less than the age of 18 years (for handguns) and domestic violence misdemeanants. Federal law does not set a minimum age requirement for the legal possession of long guns (ie, rifles and shotguns).³

Although the federal firearm prohibitions apply minimum standards for all US states, many states have enacted broader disqualifications for firearm possession including: a minimum age of 21 for all guns; convictions for some misdemeanour crimes involving violence, firearms or drugs; multiple convictions for alcohol-related offences; or convictions for serious crimes committed as a juvenile.⁴

Research supports the underlying premise of laws that widen exclusionary criteria for firearm possession: that some groups have higher rates of criminal offending than do those without a criminal history or other indicia of risk.^{5–9} For example, Wintemute and colleagues found that individuals denied legal handgun purchase, as a result of a new California law expanding firearm prohibitions to include misdemeanants convicted of crimes of violence, were less likely to commit a new crime of violence than were demographically-matched Californian misdemeanants who had been approved for handgun sales during the years just prior to the new restrictions.⁹ A study of homicide offenders in Illinois found that 42% would have been prohibited from possessing firearms as a result of a prior felony conviction; however, convictions for misdemeanours as an adult or more serious crimes as a juvenile were not reported.⁶

Under federal law, persons buying guns from licensed gun dealers must undergo a criminal history background check.¹⁰ But federal law and the law of most states do not require firearm sellers who are not licensed gun dealers to verify that purchasers of firearms are legally qualified to possess a firearm such as through a background check.⁴ Understanding how those with and without a criminal history acquire guns can also inform policies intended to keep guns from prohibited persons.

Prior research on firearm acquisition suggests that incarcerated adults often obtain their guns from casual sources such as from friends and family members, and 'off the street'.^{11–13} To our knowledge, whether and to what extent the source varies based on the legal status of the purchaser has not been investigated.

Therefore, the goals of the current study are to: (1) identify the proportion of state prison inmates

incarcerated for gun-related offences in states with the least strict standards for firearm purchase and possession who would have been prohibited from possessing firearms if laws in their states had included additional exclusion criteria and (2) describe how these inmates acquired their firearms.

METHODS

Data

This study used data from the most recent (2004) Survey of Inmates in State Correctional Facilities (SISCF), a nationally-representative survey of state prison inmates administered by the Bureau of the Census for the US Department of Justice.¹⁴ The 2004 SISCF consisted of computer-assisted personal interviews conducted between October 2003 and May 2004. Inmates were asked about a broad range of topics including: demographic characteristics; offences for which they were currently serving time; prior criminal history; gun possession and use; prior drug and alcohol use and treatment; and physical and mental health status. In the 2004 survey, 14 499 inmates were interviewed. Of those eligible to participate in the study, 89.1% participated.

Additional information about data collection and analysis methodology for the SISCF is available from the University of Michigan's Inter-university Consortium for Political and Social Research.¹⁵ Prior research using data from the SISCF include studies on incarcerated women, veterans and parents.^{16–18} No reported studies have used SISCF data on inmates who used firearms in their most recent crimes.

Study sample

To focus on the potential effects of broadening state laws regarding firearm restrictions, we limited our analysis to offenders currently serving time for an offence committed with a firearm in states that, as of 2004, did *not* have laws prohibiting persons in the following five groups from purchasing or possessing a firearm: (1) persons less than 21 years of age; (2) persons convicted of a serious juvenile offence; (3) violent misdemeanants; (4) drug misusers; and (5) alcohol abusers. To identify states meeting these criteria, we consulted the Bureau of Justice Statistics 2004 Survey of State Procedures Related to Firearm Sales,¹⁹ supplemented by legal research to confirm some state laws.

Because domestic violence misdemeanants are already prohibited from purchasing or possessing firearms under federal law,^{20–22} we included states with laws that prohibited domestic violence misdemeanants if the states did not also prohibit other violent misdemeanants from purchasing or possessing firearms. In addition, although federal law restricts firearm purchase or possession for drug misusers, the law's definition of a drug misuser does not provide objective criteria that can be implemented via a background check, limiting its practical use.²³ We excluded states with separate legal restrictions on possession of firearms by those convicted of serious offences, not technically classified as felonies, when committed by a juvenile.

Nine states—Arkansas, Idaho, Louisiana, Michigan, Mississippi, Montana, New Hampshire, Vermont and Wyoming—lacked all five types of expanded firearm disqualifications. Four additional states—Georgia, Maine, New Mexico and Wisconsin—lacked these expanded disqualifications with some exceptions. For example, New Mexico had a minimum age law stating that handgun possession is unlawful by persons <19-years-old²⁴ and Wisconsin restricted individuals convicted of a felony as a juvenile only if the offence occurred on or after 21 April 1994.²⁵ We excluded a total of 12 cases meeting these exceptions, because they were already prohibited from firearm

purchase and possession under state law. The final sample consisted of 13 states, though there were no inmates meeting our case definition in two states (New Hampshire, Wyoming; see table 1).

Measures

To determine whether offenders had a firearm while committing the crime for which they were currently incarcerated, SISCF interviewers asked, 'Did you use, carry or possess a weapon when the (...offense...) occurred?' If the answer was 'yes,' the interviewer asked, 'What kind of weapon was it?' Offenders who said they used a firearm were included in our analyses. Offenders who reported using a firearm in their current crime were asked follow-up questions, including questions about the type of gun(s) (eg, handgun, shotgun, rifle), how and where they obtained the gun, whether they fired it, and their reasons for having it.

SISCF interviewers also asked the offenders a series of questions about their prior arrests and convictions leading to probation or incarceration. Those who had been convicted and sentenced to probation or incarceration were asked about the type of offence, length of sentence, and whether they were sentenced as a juvenile or as an adult for up to 10 prior probations and 10 prior incarcerations. Offence information for juvenile convictions leading to probation and no incarceration was not collected in the SISCF.

To examine the potential for current and expanded disqualifications to curtail gun crime, we categorised offenders into the following groups based on their prior criminal convictions: (1) those who would have no firearm disqualification even under stricter state laws (described below); (2) those who were disqualified under current federal law; and (3) those who were legal firearm possessors under current federal law, but who would have been prohibited in states with stricter standards.

We further categorised offenders in the third group—those who might be impacted if the laws in their states were changed—based on whether they fell into any of the following categories: (1) age 18–20 years at incarceration for their current offence if that offence involved a handgun; (2) less than age 21 years at incarceration for their current offence if that offence involved a long gun; (3) committed a prior serious crime as a juvenile (<18-years-old); (4) conviction for a violent or firearms-related misdemeanour; (5) convictions for *two or more* drug-related misdemeanours; and (6) convictions for *two or more* alcohol-related misdemeanours. These laws were chosen because each is in effect in at least some states.¹⁹ Violent and firearm-related misdemeanours included convictions for a simple assault or a weapons offence. Drug-related misdemeanours included convictions for driving under the influence of drugs, possession or use of marijuana and unspecified drug-related offences (but did not include drug-related offences involving heroin, powder cocaine or crack cocaine which are generally felonies). Alcohol-related misdemeanours included DUI/DWI convictions or convictions for public drunkenness.

Analysis

We first calculated the proportion of offenders who would have been legally prohibited from purchasing or possessing firearms if their states had a variety of stricter laws. We then examined the method and source of firearm acquisition for offenders and calculated χ^2 statistics to identify any significant differences between offenders who were currently prohibited versus offenders who were not prohibited from purchasing and possessing firearms.

Table 1 Demographic and offence characteristics of state prison inmates incarcerated for an offence committed with a firearm in 13 states (n=253)

	n (%)
Demographic characteristics	
Sex	
Male	234 (92.5)
Female	19 (7.5)
Age when sentenced for current offence (years)	
14–17	48 (19.0)
18–20	58 (22.9)
21–24	46 (18.2)
25–29	35 (13.8)
30 and older	66 (26.1)
Race/ethnicity	
Non-Hispanic Black	169 (66.8)
Non-Hispanic White	63 (24.9)
Hispanic	9 (3.6)
Other	12 (4.7)
Education (n=251)	
Less than high school	185 (73.7)
High School or equivalent	41 (16.3)
More than high School	25 (10.0)
Marital status (n=252)	
Never married	177 (70.2)
Divorced/separated/widowed	48 (19.1)
Married	27 (10.7)
Employed in the month before incarceration (n=246)	
Full-time	129 (52.4)
Part-time/occasional	24 (9.8)
Unemployed: looking for work	32 (13.0)
Unemployed: not looking for work	61 (24.8)
State of current offence	
Arkansas	21 (8.3)
Georgia	64 (25.3)
Idaho	5 (2.0)
Louisiana	39 (15.4)
Maine	1 (0.4)
Michigan	67 (26.5)
Mississippi	27 (10.7)
Montana	5 (2.0)
New Hampshire	0 (0)
New Mexico	13 (5.1)
Vermont	1 (0.4)
Wisconsin	10 (4.0)
Wyoming	0 (0)
Current offences* †	
Violent offences	
Murder/voluntary non-vehicular manslaughter	86 (34.0)
Robbery	75 (29.6)
Aggravated assault/assault on police officer	32 (12.6)
Other violent acts	6 (2.4)
Property offences	
Burglary	6 (2.4)
Other property offences	3 (1.2)
Drug offences	
Trafficking	15 (5.9)
Possession or use	7 (2.8)
Public order offences	
Weapons offences	19 (7.5)
Parole/probation violation or contempt	2 (0.8)
Other public order offences	2 (0.8)

Continued

Table 1 Continued

	n (%)
Type of gun used in current offence‡	
Handgun	204 (80.6)
Rifle	30 (11.9)
Shotgun	25 (9.9)
Other firearm	4 (1.6)

*For inmates currently incarcerated for more than one offence, only the most serious is included here.

†All offence categories include attempted and completed offences.

‡Percentages do not sum to 100 because 10 respondents used more than one type of gun in their current offence.

RESULTS

The overall SISCf sample of 50 states included 14 499 inmates, 2046 of whom used a gun in the crime for which they were incarcerated. The distribution of the total sample of gun users was similar to the 13 states in our sample with regard to crime type, type of gun, sex, education, marital status and employment status. Our 13-state sample had a somewhat higher proportion of younger (age 14–17 years) and non-Hispanic Black offenders than for all 50 states.

Sample characteristics

Our initial sample consisted of 281 offenders who were incarcerated for offences involving firearms from the 13 states with the most lenient firearm restrictions (no stricter than existing federal law). Due to missing or insufficiently specific information about the nature of the prior convictions, 28 offenders were excluded from the analyses for a final sample of 253. The majority of the respondents came from Georgia, Louisiana, Michigan, Mississippi and New Mexico. Some of the more populous US states (eg, California, New York, Texas) were excluded from our analysis because they did not meet our legal inclusion criteria.

Three-quarters (n=190) of offenders committed their current offence (ie, the offence for which they were serving time when the interview occurred) in their state of residence. All offenders were sentenced as adults and age at sentencing for the current incarceration ranged from 14 to 55 years with a mean of 25 years. A majority of the offenders were male subjects, non-Hispanic Black, had not completed high school, were employed in the month before they were incarcerated and had never been married (table 1).

Current offences

More than three-quarters (n=199) of the offenders were serving time for a violent offence at the time of the SISCf interview. In all, 43% of these violent offenders were incarcerated for an attempted or completed murder, or voluntary non-vehicular manslaughter (table 1). The remainder of the sample was incarcerated for property, drug or public order offences (all involving firearms).

Although fewer than half (44.3%) of the offenders reported that they fired a gun while committing the current crime, most (83.4%) identified one or more other or additional reasons for possessing the gun, including using the gun to scare the victim(s) (42.7%), or for self-protection (32.4%).

Legal status for firearm possession prior to firearm offence leading to current incarceration

Inmates were categorised into three mutually-exclusive groups based on their actual or potential legal status for firearm possession (table 2). In all, 31% (n=78) of offenders would not

Table 2 Firearm prohibition status of state prison inmates incarcerated for offence committed with firearm in 13 states (n=253)

	n (%)
May possess even under stricter standards	78 (30.8)
No prior arrests or convictions and offender age ≥ 21 years	28 (11.1)
Prior arrests but no convictions and offender age ≥ 21 years	34 (13.4)
Prior non-disqualifying misdemeanour convictions, and no convictions for serious juvenile offence, and offender age ≥ 21 years	16 (6.3)
Prohibited under current state or federal laws	102 (40.3)
Prior adult (≥ 18 years) felony conviction(s) or dishonourable discharge	69 (27.3)
Offender age < 18 years at sentencing and used handgun in current offence	33 (13.0)
Would be prohibited only under stricter standards*	73 (28.9)
Handgun offender age 18–20 years at sentencing for current offence	43 (17.0)
Long gun offender age 1–20 years at sentencing for current offence	17 (6.7)
Prior conviction for serious juvenile offence	13 (5.1)
Prior conviction for firearms or violent misdemeanour	9 (3.6)
Prior conviction for 2+ drug misdemeanours	2 (0.8)
Prior conviction for 2+ alcohol misdemeanours	1 (0.4)

*These subcategories are *not* mutually exclusive.

have been disqualified from firearm possession based on prior convictions or minimum age even if their states had laws prohibiting the legal purchase and possession of firearms by persons < 21 -years-old, persons with a conviction for a serious juvenile offence, violent misdemeanants, and drug and alcohol misusers.

In the second group, 40% (n=102) of offenders were already prohibited from legal firearm possession under current state or federal law and, thus, would be unaffected by the implementation of the stricter firearm prohibition standards we considered.

The third group consists of 73 offenders (28.9%) who were not prohibited under current standards, but would have been prohibited if their states adopted stricter standards similar to those already in place in a number of other states. Most of this group (58.9% and 17.0% of all firearm offenders, n=43) would have been prohibited if their state had a law that raised the minimum age to possess a handgun to 21 years. An additional 17 offenders would have been prohibited if their state passed a law restricting possession to *all* firearms, including long-guns, for persons < 21 years. If persons convicted of a serious crime as a juvenile were to become prohibited, it would have been illegal for 13 offenders (5.1% of all firearm offenders) to purchase or possess a firearm. Nine offenders (3.6% of all firearm offenders) would also have been disqualified if their states had prohibited persons convicted of a violent or firearms-related misdemeanour from purchasing or possessing a firearm. Two offenders would have been prohibited if states were to restrict firearm purchase and possession for those with two or more drug-related misdemeanours and one offender would be prohibited if the same restriction were applied to alcohol-related misdemeanours.

How and where criminals obtained their firearms

About eight of every 10 offenders reported using a handgun (vs rifle or shotgun) in the offence for which they were serving time. Half of the offenders reported that they had bought the gun used in the crime (table 3). The second most common method of gun acquisition—cited by fewer than one in five offenders—was borrowing or holding the gun for someone. Regardless of how they obtained the gun, friends and family members were the most common source (34.0%), followed by drug dealers or other black market sources (30.4%). Only 13.4% got the gun directly from a gun store or pawnshop where federal law requires

prospective firearm purchasers to pass a background check. It is important to recognise, however, that table 3 represents only the most recent acquisition of a specific gun: it does not indicate whether the gun *ever* passed through a particular distribution channel (eg, a gun show).

There were few differences between the groups of offenders with regard to how and where they got the gun used in their most recent offence. More than half (55.6%) of offenders for whom firearm purchase and possession was legal under current standards (adding the 45 inmates who would be legal even under stricter standards with the 39 inmates who would be prohibited only under stricter standards) bought or traded for the gun used in their most recent crime compared with two-fifths (39.2%) of offenders who were prohibited under current state or federal law ($\chi^2=6.56$; $p \leq 0.01$). Offenders who were prohibited from purchasing and possessing a gun under current law acquired their gun from a licensed dealer, where a background check would be required, five times less often than offenders who were not prohibited (3.9% vs 19.9%; $\chi^2=13.31$; $p \leq 0.001$). Similarly, nearly all (96.1%) offenders who were legally prohibited from possessing a firearm acquired their gun from a supplier not required to conduct a background check.

DISCUSSION

Our findings indicate that 40% of offenders incarcerated for committing crimes with a gun in the 13 US states with the least strict standards for legal firearm purchase and possession were in possession of the gun illegally. If these states had adopted more restrictive standards like those in place in a number of other states, an additional 29% of the persons incarcerated for committing a crime with a firearm would have been legally prohibited from possessing a firearm at the time of their current offence. The vast majority of these individuals—nearly a quarter of the entire sample of firearm offenders—would have been prohibited if the minimum legal age for possessing any type of firearm was 21 years. An additional 9.9% would have been legally prohibited from firearm possession as a result of convictions for serious crimes as a juvenile or for misdemeanours involving violence, firearms, drugs or alcohol.

Nearly one in five offenders was < 18 -years-old at the time they were sentenced for the current offence; 41.9% were less than age 21 when sentenced. An even greater proportion would

Table 3 Source of gun used in current offence by state prison inmates incarcerated for offence committed with firearm in 13 states, by firearm prohibition status*

	Total (n=253) N (%)	Legal even under stricter standards (n=78) n (%)	Prohibited under current state or federal law (n=102) n (%)	Would be prohibited only under stricter standards (n=73) n (%)
How gun was got				
Stole	8 (3.2)	0 (0)	4 (3.9)	4 (5.5)
Borrowed	44 (17.4)	12 (15.4)	17 (16.7)	15 (20.6)
Bought/traded	124 (49.0)	45 (57.7)	40 (39.2)	39 (53.4)
Given as gift	21 (8.3)	8 (10.3)	9 (8.8)	4 (5.5)
Other	23 (9.1)	4 (5.1)	13 (12.8)	6 (8.2)
Don't know (DK)/refused	33 (13.0)	9 (11.5)	19 (18.6)	5 (6.9)
Where gun was got				
Gun store or pawnshop	34 (13.4)	24 (30.8)	4 (3.9)	6 (8.2)
Gun show	1 (0.4)	0 (0)	0 (0)	1 (1.4)
Friend/family member	86 (34.0)	25 (32.1)	35 (34.3)	26 (35.6)
Street/black market	77 (30.4)	14 (18.0)	36 (35.3)	27 (37.0)
Burglary	1 (0.4)	0 (0)	1 (1.0)	0 (0)
Other	21 (8.3)	6 (7.7)	8 (7.8)	7 (9.6)
DK/refused/skipped†	33 (13.0)	9 (11.5)	18 (17.7)	6 (8.2)

*If inmate used more than one gun in current offence, response pertains to the most recently acquired gun.

†Respondents who refused to disclose how they got the gun were not subsequently asked where they got it.

have fallen into the <18 group if we had data on offenders' age at the time the offence occurred rather than age at incarceration. These findings underscore the importance of minimum-age restrictions for firearms possession and disqualifications for serious offences committed as juveniles, even if the duration of these disqualifications is limited.

It is also important to consider the political feasibility of any new restrictions on access to firearms. In a 1998 survey, a large majority of respondents—including the majority of gun owners—favoured laws that would restrict guns from various categories of misdemeanants including assault and battery without a lethal weapon or serious injury, driving under the influence of alcohol, and carrying a concealed weapon without a permit.²⁶ Although public support was strong for a variety of firearm laws, firearm restrictions based on criminal history may be among the most politically feasible.^{23–27} Each firearm policy considered in this study is currently law in at least some states.

Although setting appropriate standards for legal firearm ownership is important, it is equally important to make sure that databases used to screen gun purchasers and ascertain legal status for gun possession are up-to-date so that prohibited individuals can be identified. For example, juvenile convictions must be recorded in an accessible database so that they are picked up in background checks in order for prohibitions for serious offences committed as a juvenile to be useful in restricting the legal purchase and possession of firearms in this high-risk group.

Relatively few offenders purchased their guns directly from licensed firearms dealers. Only 3.9% of individuals disqualified based on current federal or state prohibitions and 3.8% who were <21-years-old at the time of their incarceration obtained their gun from a licensed firearms dealer. Presumably most, if not all, of these prohibited individuals purchased their firearm prior to becoming a prohibited person. Among individuals who appeared to be legally qualified to purchase firearms, only one in five (19.9%) obtained their firearm directly from a licensed firearm dealer, perhaps to avoid having their firearms transactions recorded and therefore traceable to the purchaser. Given offenders' preferences for new firearms,^{13–28} it is noteworthy how criminals avoid the regulated gun market of licensed sellers and prefer the largely unregulated market involving unlicensed sellers where new guns may be harder to obtain. The lack of regulation of firearm sales by unlicensed sellers is likely to

significantly limit the government's ability to keep firearms from prohibited individuals.²⁸ Requiring all gun sales to be subject to a background check, and holding sellers accountable for failure to do so, are policies that could address this problem.²⁹

To our knowledge, this is the first study to use data on gun offenders' age and criminal histories to examine the potential benefits of strengthening the criteria for legal firearm possession. Nonetheless, it is subject to several limitations. The data used in this analysis come from inmates' self-report. As such, they share the limitations inherent to all self-report data (eg, recall and social desirability bias). And although the data were drawn from a nationally-representative survey of state prison inmates, they are not necessarily representative of state prison populations. In addition, the 13 states in our sample may not have the same distribution of offenders as in all 50 states. For example, the five states with the most offenders in our sample may be more urban, on average, than the USA as a whole. We chose states for inclusion in the sample based on their laws in 2004, the year the SISC survey took place. These laws may be different from the laws that were in effect at the time the offenders were convicted for their prior offences, though it is rare for laws prohibiting certain persons from owning guns, based on criminal history, to be repealed. Moreover, we were unable to determine whether the guns used in the current crimes were obtained in the state in which the crime was committed. This is particularly relevant for considering criteria for firearm purchase rather than possession.

The numbers of offenders with prior misdemeanour convictions are likely undercounted because we did not have status information about juveniles sentenced to probation nor did we have information about persons who were convicted but not sentenced to probation or incarceration (eg, those sentenced only to pay a fine). It is also possible (though unlikely) that some of the offenders with a prior felony had their gun rights reinstated. Finally, it is also important to remember that this is a prison population. As such, our findings may not generalise to offenders who avoid imprisonment.

However, our sample comes from a large national survey of state prison inmates and contains extensive information on their prior criminal history. In addition, we have focused on the population that is most likely to be affected by the policy changes we considered by including only offenders who used a firearm in their current offence.

What is already known on the subject

- ▶ Guns in the hands of high-risk individuals present a serious threat to public safety.
- ▶ Among high-income countries, the USA is unique in its extraordinarily high rate of firearm homicides.
- ▶ US federal law establishes minimum criteria for who may legally purchase and possess firearms; state laws vary widely in this regard.

What this study adds

- ▶ This study is the first to use data on incarcerated gun offenders' age and criminal histories to examine the potential benefits from strengthening the criteria for legal firearm possession.
- ▶ Nearly three of every 10 gun offenders in the 13 US states with the least stringent criteria for legal gun ownership would have been prohibited from purchasing or possessing a firearm when they committed their most recent offence if their states had more restrictive laws in place.
- ▶ Offenders for whom access to firearms was legal under current standards were five times more likely to have obtained their gun from a gun store or pawnshop than were offenders who were prohibited under current state or federal law.

Our findings indicate that stricter gun ownership laws in states with the lowest standards would have made firearm possession illegal for many who used a gun to commit a crime. We are uncertain about the degree to which stricter legal standards for firearm possession might deter criminal gun possession and use. But, adding barriers for the acquisition of guns by high-risk persons is an underused potential intervention.

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REFERENCES

1. **Centers for Disease Control and Prevention.** Web-based injury statistics Query and reporting System (WISQARS). National Center for Injury Prevention and Control, Centers for Disease Control and Prevention (producer), 2007. <http://www.cdc.gov/ncipc/wisqars> (accessed 10 Nov 2011).
2. **Richardson EG,** Hemenway D. Homicide, suicide, and unintentional firearm fatality: comparing the United States with other high-income countries, 2003. *J Trauma* 2011;**70**:238–43.
3. **18 U.S.C. §922(g).** 2011.
4. **Vernick JS,** Webster DW, Vettes KA. Law and policy approaches to keep guns away from high risk people. In: Culhane JG, ed. *Social Issues, Welfare Consequences, and Public Health Law*. New York: Cambridge University Press, 2010.
5. **Berk R,** Sherman L, Barnes G, et al. Forecasting murder within a population of probationers and parolees: a high stakes application of statistical learning. *J Roy Stat Soc* 2009;**172**:191–211.
6. **Cook PJ,** Ludwig J, Braga AA. Criminal records of homicide offenders. *JAMA* 2005;**294**:598–601.
7. **Huebner B,** Varano S, Barnes G, et al. Gangs, guns, and drugs: recidivism among serious young offenders. *Criminol Publ Pol* 2007;**6**:187–222.
8. **Lucker GW,** Holt VL, Kruzich DJ, et al. The prevalence of antisocial behavior among U.S. Army DWI offenders. *J Stud Alc* 1991;**52**:318–20.
9. **Wintemute GJ,** Drake CM, Beaumont JJ, et al. Prior misdemeanor convictions as a risk factor for later violent and firearm-related criminal activity among authorized purchasers of handguns. *JAMA* 1998;**280**:2083–7.
10. **18 U.S.C. §922(s).** 2011.
11. **Cook PJ,** Ludwig J, Venkatesh S, et al. Underground gun markets. *Econ J* 2007;**117**:F588–618.
12. **Wright JD,** Rossi PH. *Armed and Considered Dangerous. Armed and Considered Dangerous: A Survey of Felons and their Firearms*. New York: Aldine de Gruyter, 1994.
13. **Webster DW,** Freed LH, Frattaroli S, et al. How delinquent youth acquire guns: initial versus most recent gun acquisitions. *J Urban Health* 2002;**79**:60–9.
14. **U.S. Department of Justice.** Bureau of Justice Statistics, *Survey of Inmates in State and Federal Correctional Facilities, 2004*. Ann Arbor, MI: Inter-university Consortium for Political and Social Research [producer and distributor], 2007. doi:10.3886/ICPSR04572
15. **U.S. Department of Justice.** Bureau of Justice Statistics, *Survey of Inmates in State and Federal Correctional Facilities, 2004*. Codebook. Ann Arbor, MI: Inter-university Consortium for Political and Social Research [producer and distributor]. <http://dx.doi.org/10.3886/ICPSR04572.v1>
16. **Glaze LE,** Maruschak LM. *Parents in prison and their Minor Children*. Washington, DC: U.S. Department of Justice, Bureau of Justice Statistics, 2008. Report No.: NCJ 222984.
17. **Leigey ME,** Reed KL. A woman's life before serving life: examining the negative pre-incarceration life events of female life-sentenced inmates. *Women Crim Just* 2010;**20**:302–22.
18. **Noonan ME,** Mumola CJ. *Veterans in State and Federal Prisons, 2004. Special Report*. Washington, DC: U.S. Department of Justice, Bureau of Justice Statistics, 2007. Report No.: NCJ 217199.
19. *Survey of State Procedures Related to Firearm Sales, Midyear 2004*. Washington, DC: National Institute of Justice, Bureau of Justice Statistics, 2005. Report No.: NCJ 209288.
20. **18 U.S.C. §922(d)(9).** 2011.
21. **18 U.S.C. §922(g)(9).** 2011.
22. **Vigdor ER,** Mercy JA. Do laws restricting access to firearms by domestic violence offenders prevent intimate partner homicide? *Eval Rev* 2006;**30**:313–46.
23. **Webster DW,** Vernick JS. Keeping firearms from drug and alcohol abusers. *Inj Prev* 2009;**15**:425–7.
24. **N.M. Stat Ann. §30-7-2.2.** 2004.
25. **Wis. Stat. §941.29.** 2004.
26. **Teret SP,** Webster DW, Vernick JS, et al. Support for new policies to regulate firearms: results of two national surveys. *N Engl J Med* 1998;**339**:813–18.
27. **Vernick JS,** Rutkow L, Webster DW, et al. Changing the constitutional landscape for firearms: the Supreme Court's recent Second Amendment decisions. *Am J Public Health* 2011;**101**:2021–6.
28. **Cook PJ,** Molliconi S, Cole TB. Regulating gun markets. *J Crim Law Criminol* 1995;**86**:59–92.
29. **Webster DW,** Vernick JS, Bulzacchelli MT. Effects of state-level firearm seller accountability policies on firearm trafficking. *J Urban Health* 2009;**86**:525–37.